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THE HISTORY OF LEPROSY.

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FEW diseases have excited the interest of the pathologist, the historian or the lay public so much as leprosy. It ranks next only to the plague among the great scourges which have afflicted the human race and left their mark upon the history, literature and imagination of the people. The peculiar phenomenon of its rise to epidemic proportions in certain parts of the world, followed by its complete disappearance from these regions, is one which has proved a favourite topic among historians and epidemiologists alike.

Where and when did leprosy have its origin, and what was the manner of its spread? Before attempting to answer such questions it is necessary to scrutinize the sources of our information regarding this disease by reference to contemporary literature, and to inquire into the history of its nosology, in order to determine how far we may be justified in accepting alleged allusions to it in ancient literature as having any genuine reference to the condition we now designate as leprosy.

Nosology.

Perhaps the earliest reference to what might have been leprosy is contained in aphorisms attributed to the legendary Chinese emperor Hoang-Ti, who is also regarded by the Chinese as the father of medicine. He is supposed to have written about the seventeenth century B.C. In the passage referred to we are told that "the disease corrodes the nasal septum, the complexion alters, the body becomes covered with sores, from which there exudes a foul

discharge". This quotation is taken from the *"Tô Vân"* of Wong Nyang, a résumé of the book attributed to Hoang-Ti, but of much more recent date, and it is possible that the original text may have been altered and that additions may have been made thereto (Barbézieux, 1914).

In the *"Rig Veda"*, the earliest of the Indian scriptures, written about 1500 to 900 B.C., there are references to a disease called *kushtah*. In one hymn it is related how a daughter of the Rishi Cakehivan and in another how Rishi Syava are said to have suffered from the disease and to have been cured by the Asvins. There is, however, no description of the condition; and the fact of its having been cured militates against the hypothesis of its being leprosy. In the *"Atharva Veda"*, which is a little more recent than the *"Rig Veda"*, the same disease is referred to in incantations for the cure of *kushtah* by a dark plant. Thus, to quote one of these incantations[1] (*"Atharva Veda"*, i, 23):

1. Born by night art thou, O Plant, dark, black, sable.
   Do thou that art rich in colour stain this leprosy and the gray spots.
2. The leprosy and the gray spots drive away from here—
   May their native colour settle upon thee—the white spots cause to fly away!
3. Sable is their hiding place, sable their dwelling place, sable art thou, O Plant:
   Drive away from here the speckled spots.
4. The leprosy which has originated in the bones and that which has originated in the body and upon the skin,
   The white mark, I have destroyed with my charm.

In the code of Manu (500 B.C. to A.D. 200), *kushtah* is made the ground for divorce, but the disease is not described. The lineaments of the disease, although obscured by other skin diseases, are also said to be discernible in the writings of Charaka (second century A.D.) and Susruta (fifth century A.D.). Significantly enough, it is regarded as a punishment for unchastity as in Gautama's commentaries on Manu.

According to some authorities, leprosy is referred to in Ebers's papyrus (1550 B.C.); but this is doubtful. Brugsches, in his *"Histoire d'Egypte"*, mentions that in a medical papyrus discovered by him at Memphis and supposed to have been composed during the reign of Husapti, fifth king of Egypt, in 2400 B.C., are given a number of receipts for the cure of a disease *"du genre de la lèpre"*; but the association with leprosy is probably mythical (Engel). The same may be said of his reference to another passage, the date of which he gives as 4200 B.C.

[1] In a note upon the above in Max Müller's "Sacred Books of the East" it is said that the practice connected with these hymns is defined by the commentators as a cure for white leprosy (*svetakushthapanadanaya*). While reciting, the priest rubbed in dung upon the spots until they became red, and smeared upon them yellow sandal and yellow turmeric. He then cut off the white scurf. While the above allusion may have referred to leucoderma or even to the maculo-anæsthetic type of leprosy, it is more probable, in view of the notes of the commentators, that in reality it referred to psoriasis, and the allusion to the bones may mean that the eruption occurred in the neighbourhood of bony prominences.

What is believed by many to be the earliest authentic description of leprosy occurs in the Old Testament, in the thirteenth chapter of Leviticus. Since the Exodus is believed to have occurred in Meneptah's time (1320 B.C.), this would bring the date of Leviticus to some time just after the fourteenth century B.C. The idea that our modern leprosy is the disease mentioned in the Bible dies hard; and since the biblical notions of leprosy have played such an important part in the history of the disease, it is necessary to examine the evidence.

Briefly, the account given in the thirteenth chapter of Leviticus is to the effect that if a man has a suspicious-looking skin disease, be it a rising or swelling (*s'eth*), a scab or gathering (*sappachath*), or a bright spot (*berat*), he must go before the priests, who examine it; and then, whatever the nature of the eruption, they apply the following general rule. If it should be pale in colour, not deeper than the skin, if the hair is not white, and if the disease does not spread in spite of treatment, then it is clean. But if it is deeper than the skin, exposing a raw surface (*chay*) and if the hair is white, or if the infection spreads in spite of treatment, then it is regarded as *tsarat*, and the sufferer is unclean. Whilst the second to ninth verses seem to apply to general rules, in the remaining parts of the chapter various skin diseases are discussed in groups. Thus verses 9 to 17 seem to apply to chronic skin diseases affecting the body surface generally; verses 18 to 28 to septic and acute inflammatory conditions, such as boils, carbuncles, and possibly erysipelas; verses 29 to 37 to infections of the beard and the scalp, such as sycosis, impetigo, and possibly ringworm; verses 38 to 46 to infections of the face, forehead and scalp. Verses 47 to 59 seem to be concerned with fungi growing in garments, and in Chapter xiv, verses 33 to 55, we hear of the term *tsarat* being applied to what may have been dry-rot, borers and mildew in the house.

Throughout, the word *tsarat* (from the root signifying sting or affliction, hence distress), translated as leprosy, refers not to a specific disease, but to a malignant form of various affections which rendered the subject unclean. Again, the expression unclean probably means untouchable, to be avoided, or ceremoniously unclean, and not necessarily infectious, as the term applies equally to boils and to women during the menstrual period; while in the New Testament we read how Jesus touched the "leper", saying "Be thou clean", thereby releasing from him the ban of ceremonial pollution, a man who had been an outcast and an untouchable pariah. It should also be noted that the passage reading "The priest shall shut up him that hath the plague seven days" does not refer to isolation (which would mean that a large number of people would have had to be shut up for long periods for trivial causes), but to treatment, and should be translated: "The priest shall bind up the sore". That is to say, "shut up" should be "bind up", "plague" (*nega*) should be "sore", and "him that hath", which is in italics in the Authorized Version, is super-

fluous. This at once renders the text intelligible. With these explanations in mind the reader may judge for himself whether the relevant passages suggest leprosy. The general rules are given in verses 2 to 8.

2. When a man shall have in the skin of his flesh a rising,[1] a scab,[2] or a bright spot[3] and it be in the skin of his flesh like the plague[4] of leprosy; then shall he be brought unto Aaron, the priest, or unto his sons, the priests.
3. And the priest shall look on the plague in the skin of the flesh; and when the hair in the plague is turned white, and the plague in sight be deeper than the skin of his flesh, it is a plague of leprosy: and the priest shall look on him and pronounce him unclean.
4. If the bright spot be white in the skin of his flesh and in sight be not deeper than the skin, and the hair thereof be not turned white; then the priest shall shut up him that hath the plague[5] seven days.
5. And the priest shall look on him the seventh day: and, behold, if the plague in his sight be at a stay, and the plague spread not in the skin; then the priest shall shut him up seven days more.
6. And the priest shall look on him again the seventh day; and behold, if the plague be somewhat dark[6] and the plague spread not in the skin,[7] the priest shall pronounce him clean: it is but a scab:[8] and he shall wash his clothes, and be clean.
7. But if the scab spread much abroad in the skin after that he hath been seen of the priest for his cleansing,[9] he shall be seen of the priest again.
8. And if the priest see that, behold, the scab spreadeth in the skin, then the priest shall pronounce him unclean: it is a leprosy.[10]

Rules regarding cutaneous diseases affecting the body surface generally are given in verses 9, 10 and 11.

9. When the plague of leprosy is in a man, then he shall be brought unto the priest;
10. And the priest shall see him: and, behold, if the rising be white in the skin, and it have turned the hair white, and there be quick raw flesh in the rising;
11. It is an old[11] leprosy in the skin of his flesh, and the priest shall pronounce him unclean, and shall not shut him up:[12] for he is unclean.

Verses 10 and 11 evidently refer to chronic white patches, with a raw-looking surface and resistant to treatment. It must be admitted that there is a certain resemblance between various scaly skin eruptions, such as psoriasis, syphilitic psoriasis, macular syphilides, *tinea circinata*, or even *lichen planus*, and the maculo-anæsthetic form of leprosy. But it is striking that there should be no mention of anæsthesia or of the terrible deformities that result from leprosy. Nor can it be maintained that the description applies only to the early stages of the disease, as we are told that it is old.

12. And if a leprosy break out abroad in the skin, and the leprosy cover all the skin of him that hath the plague from his head even to his foot, wheresoever the priest looketh;

13. Then the priest shall consider: and, behold, if the leprosy have covered all his flesh, he shall pronounce him clean that hath the plague: it is all turned white: he is clean.
14. But when raw flesh appeareth in him, he shall be unclean.
15. And the priest shall see the raw flesh, and pronounce him to be unclean: it is a leprosy.
16. Or if the raw flesh turn again, and be changed unto white, he shall come unto the priest;
17. And the priest shall see him; and, behold, if the plague be turned into white; then the priest shall pronounce him clean that hath the plague: he is clean.

It hardly seems likely that the above should refer to a generalized macular leprosy, breaking down with the appearance of raw flesh and then undergoing healing. Verses 13 and 14 might refer to ichthyosis, but the subsequent verses might be interpreted as having reference to some condition like *pityriasis rubra* supervening upon a preexisting generalized eruption, such as scaly eczema, psoriasis, *lichen planus* or ichthyosis, then disappearing, to leave signs of the original milder affection.

38. If a man or a woman have in the skin of their flesh bright spots,[1] even white bright spots;
39. Then the priest shall look: and, behold, if the bright spots in the skin of their flesh be darkish white; it is a freckled spot[2] that groweth in the skin; he is clean.

This condition evidently refers to a benign type of eruption characterized by dull white patches. Have we here any reason to identify it with macular leprosy? Might it not have been leucoderma or, as the face and head seem to have been under discussion, *lupus erythematosus* or a mild form of psoriasis?

42. If there be in the bald head, or in the bald forehead, a reddish sore; it is a leprosy sprung up in his bald head, or his bald forehead.
43. Then the priest shall look upon it; and, behold, if the rising of the sore be white reddish in his bald head, or in his bald forehead, as the leprosy appeareth in the skin of the flesh;
44. He is a leprous man, he is unclean: the priest shall pronounce him utterly unclean, his plague is in his head.
46. All the days wherein the plague shall be in him he shall be defiled.

A reddish-white rising or swelling occurring on the forehead, or on top of the forehead or head, from which the hair disappeared, was the most unclean disease of all. It has been supposed that here we have a picture which may pass for tuberculoid leprosy. It may be pointed out, however, that while the falling out of the eyebrows may be an early sign of leprosy, baldness is not a feature of tuberculoid leprosy, and lepra bacilli seldom attack the scalp. The condition referred to might be favus, a sufficiently revolting disease, or ringworm, and it is to be noted that it is implied in verse 46 that the condition is not incurable.

It must, therefore, be admitted that we have no clear evidence that the disease now called leprosy is included in the list of affections described in Leviticus. The same may be said of the leprosy of Miriam (Numbers xii, 10), or that of Naaman (2 Kings, v, 1) and Gehazi (2 Kings, v, 27), which

[1] *S'eth.*
[2] *Sappachath.*
[3] *Berat.*
[4] *Nega*—a sore resembling or suspected of being an ulcerous sore.
[5] Bind up the sore.
[6] *Metrah*—dim, faded.
[7] That is, response to treatment.
[8] *Sappachath.*
[9] In spite of treatment.
[10] *Tsarat.*
[11] Chronic.
[12] That is, to bind up would be futile, as the condition is not amenable to treatment.

[1] *Berat.*
[2] *Boak.*

is merely described as being "white as snow", just as in the case of Moses the hand became "leprous as snow". It evidently refers to a white scaly or a macular eruption like psoriasis, or to white patches like leucoderma. At all events the leprosy of Naaman did not prevent him from living at court and discharging his duties as general of the army; nor was Gehazi's health sufficiently impaired to debar him from acting as attendant to Elisha and holding intercourse with the king.

FIGURE I.
Procession of lepers at Amsterdam in 1608. (After Visscher, *Aesculape*, 1929.)

Of the leprosy of Uzziah (2 Chronicles, xxvi, 19-21) it is merely stated that it first appeared on the forehead and that he was a leper to the day of his death, but no details are given. The disease of Job has been held by some to be leprosy, an opinion

FIGURE II.
Lazarus the leper at the palace of the wicked rich man. Window glass of sixteenth century. (*Aesculape*, August, 1929.)

which dates back to Origen; but it can hardly be said that the "boils" and intolerable itching which caused Job to sit down among the ashes, using a potsherd to scrape himself with, is a feature of any form of leprosy. Rather do we think of *dermatitis herpetiformis*. That the disease is said to "touch his bone and his flesh" need not be taken to indicate deep-seated ulceration, but, as may be gathered from Chapter xix, verse 20 ("My bone cleaveth to my skin and to my flesh"), emaciation which, with sleeplessness, exhaustion and mental disturbance

FIGURE III.
Leper ringing his bell. (After Van Orley, *Aesculape*, August, 1929.)

(all of which are features of Job's disease), are well-known complications of *dermatitis herpetiformis*.

Leprosy is not described in the New Testament, but the word λεπρα, used by Saint Luke, himself a

FIGURE IV.
Saint Wandrille, Abbot of Fontenelles in the County of Caux, instructing a leper. (After Hans Burgmaier, *Aesculape*, August, 1930.)

physician, almost certainly refers to the disease known contemporaneously among the Greeks by that name; that is, various kinds of scaly eruption, such as psoriasis, and not true leprosy. Hippocrates (460 B.C.) did not describe elephantiasis, which is generally admitted to have been leprosy; but he

alluded to various scaly cutaneous affections under the term λεπραι (λεπος, a husk or scale), of which there were several varieties, such as αλφος, μελας and λευκη. Λευκη he speaks of as the Phœnician disease, and αλφος he regards as a blemish rather than a

FIGURE V.
Saint Peter healing a leper. (After A. Dürer, *Aesculape*, August, 1930.)

disease. These diseases are also mentioned by Galen (A.D. 131-201); but it is to Celsus (A.D. 30) that we have to turn for an explanation of the Greek terms.

The λεπρα of Hippocrates was the *vitiligo* of Celsus, and evidently refers to different varieties of psoriasis or other scaly skin eruptions. In λεπρα αλφος the efflorescence is white, of slight roughness

FIGURE VI.
Lepers appealing for alms. (The work of an unknown painter of the Haarlem school.) (*Janus*, 1919.)

and not continuous, but occurs as if in dispersed spots, though occasionally these are broad and extensive, and it always creeps over the body with a healthy-looking surface intervening. Λεπρα μελας differs from the αλφος merely in colour, since it is in some cases brownish, giving to the skin an appearance as if a shadow were cast over it. Λεπρα λευκη has something in common with αλφος; but it is whiter and affects the skin, and the hair becomes white like wool. Λεπρα λευκη is described as the most malignant variety, and some have thought to identify it with true leprosy. It is described as follows:

> Scales on an elevated base, which are glossy white, with a deep central depression, encircled with a red border; patches increasing in size; hairs on the patches, white or hoary, diffused over the body, and contagious.

FIGURE VII.
Leper house. (Manuscript illumination from Vincent de Beauvais, thirteenth century.)

But it is no more possible to say of this that it refers to leprosy than it is of the account given of cutaneous diseases in Leviticus. There is the same absence of reference to deformities and to anæsthesia.

Attempts have been made by Good, Belcher (1869), Shapter (1834), Bennett (1887) and others

FIGURE VIII.
Attending the sick in a monastic hospital.

to identify the various varieties of skin diseases mentioned by Hippocrates and described by Celsus with those mentioned in Leviticus, in spite of the separation in time of the two accounts by 1,000 to 1,500 years. Thus *berat*, or bright spot, λεπρα and *vitiligo* are regarded as synonymous general terms covering three varieties of disease, namely: *boak*,

the benign variety corresponding to λεπρα αλφος; *berat kehah*, corresponding to λεπρα μελας (the dark but benign variety referred to in Leviticus xiii, 6); and *berat lebena*, corresponding to λεπρα λευκη (the malignant variety referred to in verses 9 and 10). The word *tsarat* is supposed to be a general term covering the two last more serious sub-varieties, namely, *berat kehah* and *berat lebena*.

These interpretations would seem unjustifiable, since, as already pointed out, the term *tsarat* is applied to a great variety of malignant skin diseases, and even to fungus growths on garments and in the house. It is definitely not applied to the dark variety (*kehah*).

What the term *satyria*, mentioned by Aristotle, means is uncertain, since he gave no details; and it is difficult to know what store is to be laid on the opinions of Rufus and Galen, writing four or five hundreds years after Aristotle, who identified it with elephantiasis or tuberculoid leprosy; nor can we be certain that Galen is right when he identifies the λεπρα λευκη with the same disease. The term *satyria* has, however, come to be applied to the satyr-like facies of tuberculoid leprosy.

It is not until we come to the writings of Aretaios, the Cappadocian, in the second and third centuries A.D., that we meet with any clear and unequivocal description of leprosy, under the title of elephantiasis, a disease of which the author was himself a victim. He gives an account of the facial appearance known as leontiasis; and in connexion with elephantiasis he remarks: "It is disgusting to the sight and terrible in all respects" (*"est visu foedus et in omnibus terribilis"*); also that the disease is as much greater than other diseases as the elephant is bigger than all other animals. It should be mentioned, however, that this explanation of the word elephantiasis is not the only possible one, as the term may have reference to the thickened and insensitive condition of the skin, causing it to resemble the hide of the elephant. A brief notice of the disease is also given by Rufus of Ephesus in the second century A.D., when he mentions that it (elephantiasis) had previously been described by Strato, the Erisistratean (third century B.C.).

Galen, the contemporary of the Indian physician Charaka, writing towards the end of the second century A.D., gives a detailed account of the disease. Then follow accounts by Oribasius in the fourth century (in which were preserved the previous descriptions by Rufus and Archigenes), and by Aetius in the sixth century, while the disease is also referred to by Paul of Ægina in the seventh century. The Arabian physicians Rhazes (860-932) and Avicenna (950-1037) likewise describe it under the title *juzam* or *judam*.

With the rise of the school of Salerno in the eleventh and thirteenth centuries and the introduction of Arabic medicine to that school by Constantinus Africanus, great confusion was introduced in the nomenclature of the disease. This arose through the Arabians having described a disease known as *das fil* or "elephant disease", our modern filarial disease, elephantiasis. This Constantinus and the translators of the Arabian physicians wrongly took to be the same as the elephantiasis of the Greek physicians Aretæus and Aetius, while the real equivalent of the Greek elephantiasis was the disease known to the Arabians as *juzam* or *judam*. The Arabian *juzam* was, however, erroneously rendered "lepra", a word applied by the Greeks, as we have seen, to various scaly eruptions. In this way the term "lepra" or "leprosy" came to be applied to the disease which the Arabians called *juzam* and which was the real equivalent of the Greek elephantiasis or true leprosy, and this nomenclature has persisted down to the present day. From this time onwards, therefore, the term leprosy is intended by all medical writers to denote the disease which we now call by that name.

During the Middle Ages, medicine, like most learning, was largely in the hands of the clergy, and there can be no doubt that the term leprosy was applied to a great many other conditions as well as to the true leprosy which was at that time prevalent. A leper was in fact one full of sores (*ulceribus plenus*), like Lazarus in the parable, and a great many other loathsome conditions were included under that rubric.

In the thirteenth century we have excellent accounts of leprosy by the monk Theodoric of Bologna, by Lanfranc of Milan, and by Bachuore of Barcelona. With the rise of the school of Montpellier in the fourteenth century a number of writers on leprosy appeared in France and England. Whilst these have copied largely from Avicenna, Rhazes and Theodoric, they have improved upon their models. The Scotsman, Bernhard Gordon, or Gordino (1308), first professor of medicine in Montpellier, divides the symptoms into three classes: the occult, the infallible, and the terminal or breaking up.

1. The occult premonitory signs are a reddish colour of the face, verging to duskiness; the expirations begin to be changed; the voice grows raucous, the hairs become thin and weakened: the perspiration and breath incline to fœtidity; the mind becomes melancholic, with frightful dreams and nightmares; in some cases scabs, pustules and eruptions break out over the whole body; the disposition of the body begins to become loathsome, but still, while the form and figure are not corrupted the patient is not to be adjudged for separation, but is to be most strictly watched.

2. The infallible signs are enlargement of the eyebrows with loss of hair, rotundity of the eyes, swelling of the nostrils externally and contraction of them within; voice nasal; colour of the face glossy, verging to a darkish hue; aspect of the face terrible, with a fixed look and with acumination and contraction of the pulps of the ears. There are many other signs, as pustules and excrescences, atrophy of the muscles, and particularly those between the thumb and the forefinger; insensibility of the extremities; fissures and infections of the skin; the blood, when drawn and washed, containing black earthy and rough sandy matter and other marks which authors prominently mention, but for me those suffice which are to be found in the face. The above are those manifest and evident signs which, when they do appear, the patient ought to be separated from the people.

3. The signs of the last stage and breaking up ("*naufragium*") of the disease are corrosion and falling-in of the cartilage forming the septum of the nose; fissure and division of the feet and hands, enlargement of the lips and a disposition to glandular swelling; dyspnœa and difficulty in breathing; a voice hoarse and barking; the aspect of the face frightful and of a dark colour; the pulse small and imperceptible.

Guy de Chauliac divides the symptoms into "univocal" and "equivocal". The univocal signs are:

(i) Rotundity of the ears and eyes; (ii) thickness and tuberosity of the eyebrows, with falling off of their hair; (iii) dilatation and disfiguration of the nostrils externally, with stricture of them within and fœtidity of the lips; (iv) voice raucous and nasal; (v) fœtidity of the breath and of the whole person; (vi) fixed and horrible satyr-like aspect.

The accounts given by Gilbert, the first English writer on leprosy, and by John of Gaddesden, professor of medicine at Merton College, Oxford, are along similar lines. John of Gaddesden thus describes the condition:

Facie, rotundatio oculorum, contractio palpebrarum, lacrimositas multa et aquositas oculorum, depilatio superciliorum et grossities eorum; dilatio narium exterius et constrictio interius, et coartatio anhelitus, quasi si cum naribus loqueretur. Et color faciei lividus vergens ad fuscedinem mortificatum. Terribilis aspectus faciei cum fixo intuitu, contractio et palpebrarum et aurium. Infectio cutis maculosa. Tuberositas et pustulae in facie et nodositas. Ista omnia et major pars sunt infallibilia signa leprae actualis.

Bartholomew Glanville says that persons afflicted with leprosy have

redde whelks and pymples in the face, out of whome oftenne runne blood and matter; en such the noses swellen, and ben [become] grete, the vertue of smellynge faylyth, and the brethe stynkyth ryght fowle,

and when the disease is advanced they are

unclean, spotyed, glemy, and quythery [watery], and the nosethrilles ben stopye, the wasen of the voys is rough and the voys is horse, and the heere falls.

Gordon, John of Gaddesden and other authors insist that no one should be judged a leper and separated from mankind until the form and figure (*"forma et figura"*) of the face are altered (*"corruptio figurae"*).

There can be no doubt as to the nature of the disease which the above authors describe, but writing at a time when leprosy was on the wane and syphilis was on the increase, it is not surprising that we should find in them many allusions which suggest confusion with syphilis.

Thus Gordon relates how a noble lady came to Montpellier to be treated by him for an alleged leprosy. The bachelor of medicine charged with her care fell in love with her *"et jacuit cum ea, et impregnavit eam, et perfectissime leprosus factus est"*.

Again, to quote Gilbert:

In hoc genere, causa est accessus ad mulierem ad quam accessit prius leprosus; et corrumpit velocius vir sanus quam mulier a leproso. Et penetrant in nervos calidos et arterias et venas viriles et inficiunt spiritus et bubones.

Many authors have written on leprosy since the fourteenth century, but few have improved upon the mediæval writers until comparatively recent times. In 1842 there appeared the great work of Danielssen and Boeck, the most complete and accurate description of the disease as a clinical entity up to that time. The final definition of the disease was achieved by the discovery of *Bacillus leprœ* by Hansen in 1874.

Origin and Spread of Leprosy.

It is evident from the above account of the history of the nosology of leprosy that we have no unchallengeable record of any description of the disease before the first two or three centuries of the Christian era. Various references to elephantiasis enable us, however, to infer that leprosy existed in Egypt and Greece for at least two or three centuries before Christ; also, that it was unknown or rare in Italy and other parts of the Roman Empire until the first century B.C. Curiously enough, the first seemingly authentic records of its existence in China appear at this period. We have seen that, according to Rufus, elephantiasis was described by Strato as far back as 300 B.C. Again, Lucretius, writing in 95 B.C., says: *"Est elephas morbus, qui propter flumina Nili gignitur Aegypto in medio, neque praeterae usquam."* ("There is a disease called elephantiasis which has its rise on the river Nile in the middle of Egypt and in no other country.")

Celsus in the first century A.D. says: *"Ignotus paene in Italia morbus est quem ελεφαντιασις Graeci vocant."* Plutarch makes the physician Artemidorus, a contemporary of Pompey, say that leprosy first became known in the Roman Empire at the time of Asclepiades, that is, in the first century B.C. Galen, again, says that in his time (that is to say, the second century):

There are in Alexandria many sufferers from elephantiasis on account of the diet and the heat of the country. On the contrary, in Germany and in Mysia this affection is very rarely seen. It is almost never seen among the Scythians, who drink milk, but in Alexandria it is common on account of the diet; there they eat much oatmeal and lentil brose, snails and salt fish, and there are even some who eat ass flesh and the like.

Apart from such references to elephantiasis, which carry us back to about 300 B.C., our knowledge of the existence and geographical distribution of leprosy in the ancient world is purely a matter of speculation. If we are to admit that the Greek λευκη does indeed refer to leprosy, then we would gather from the statements of Pausanius and Herodotus that it existed in Persia in the fifth and sixth centuries B.C. Herodotus, writing in the sixth century B.C., makes the following remarks:

A citizen infected with leprosy properly so-called, or with the species of leprosy known as λευκη, is not permitted to enter the town or to have any communication with the Persians. It is, according to them, a proof that they have sinned against the sun.

Similarly, we would conclude from Hippocrates that it was present in Greece in the fifth century B.C. If we were to accept the allusion contained in the ancient scriptures and papyri as having reference to true leprosy, then it would be necessary to suppose that leprosy must have existed as an endemic disease in Egypt, India, China and Japan,

for at least fifteeen hundred years B.C., and possibly from time immemorial. Enthusiastic medical palæontologists and anthropologists would even carry us further back. In excavations at Solutré (Saône-et-Loire) human remains have been discovered which are said to exhibit bony lesions characteristic of leprosy (Vignard, 1884). Again, Labourt (1854), from researches in comparative philology and from the examination of megalithic monuments in Brittany, finds evidence of the existence of leprosy among the primitive inhabitants of Gaul. It should be mentioned that according to some authors Ethiopia, where the disease to this day is rife among the negro population, is the true cradle of leprosy, and that the disease spread as the result of the extension of the Egyptian Empire under Thothmes (eighteenth dynasty), who antedated Moses by more than a century, or as a result of the colonization of Ethiopia, which began in the twelfth dynasty. According to Munro the first account of leprosy in Egyptian records dates back to the reign of Rameses II (1350 B.C.). In these mention is made of its having occurred among the negro slaves from the Sudan and Darfur, but the reading is doubtful.

In the absence of any definite evidence as to the existence of leprosy in remote antiquity, it is impossible to say when or where the disease originated, whether in a single focus such as Egypt or in widely separated endemic foci throughout the world. The epidemic prevalence of the disease may have been determined from time to time by the development of the necessary social and biological conditions. Changes in social habits and the existence of poor dietetic or housing conditions generally may have played their part; but among the factors predisposing to epidemics is one which has been frequently overlooked, namely, the movements of peoples—either uninfected people coming into an area in which the disease, after a period of long endemicity, had become milder or was disappearing, or the invasion of an uninfected people by an infected one. In this light we have to regard the effect of the spread, both in prehistoric and historic times, of various Asiatic and northern European people southward and westward, where they may have become infected by the autochthonous population: the northward spread of Roman civilization, the movements of trading peoples like the Phœnicians, and in more recent times the spread of European peoples to the New World and Australia. Since leprosy is a disease which is communicated by close and prolonged contact, it is rather the spread of peoples than the transient occupation of countries by invading armies which would be important; and we need not, therefore, accept such facile explanations of its spread as, for example, that leprosy was carried to India by the armies of Alexander, to Greece by those of Xerxes, or to Italy by the soldiers of Pompey returning from Greece. On the other hand, the transference of captives and slaves or the prolonged occupation of territories, such as occurred with the successive rises of the Persian Empire under Cyrus, Cambyses and Darius, of the Macedonian Empire under Alexander, or of the Roman Empire, all of which in turn occupied Egypt and the Levant, would be of more importance and may have helped to convey the disease to Greece and Rome. Certainly there were plenty of foreigners, slaves, captives and traders, in Rome when leprosy first became known there in the first century B.C. The Roman armies, again, owing to their prolonged occupation of European territory, together with the fact that they were often transferred from the east and that Roman civil officers accompanied the army, may have helped to disseminate it. With regard to the Crusades, it is certain that leprosy was common and that leper houses were being founded in various parts of Europe before the first band of Crusaders set off. While we know that a certain number of Crusaders became infected, including various noble persons, such as Baldwin I of Jerusalem, it is improbable that the Crusaders played any very important part in the spread of leprosy, except perhaps indirectly through the devastating effect they had upon social conditions in Europe. It has been suggested that the real disease they were responsible for spreading was syphilis, which became very common just towards the end of the Crusades.

Leprosy, which, as we have seen, was known in Italy in the first century B.C., became diffused throughout Europe after the beginning of the Christian era, rising to epidemic proportions in the Middle Ages and then gradually declining. The evidence for this is to be found in the writings of the chroniclers and in numerous edicts, laws and regulations relating to lepers and institutions for their care. There can be no doubt that these institutions were founded only after leprosy had become prevalent, while their number cannot be taken as an accurate index of the number of lepers, but rather of the religious zeal which prompted action on behalf of "Christ's poor". There is a doubtful reference to leprosy having been reported in Spain and in England (Barton Burke) as early as 60 B.C.; while we gather from Galen that it was present, although rare, in Germany in the second century A.D. It is recorded in Ireland during the fifth century and in Scotland in the seventh century. During the Roman occupation it was gradually increasing in England. In the fifth and sixth centuries it occurred in Spain when that country was being overrun by the Vandals and Vizigoths. In the sixth and seventh centuries leper houses were being founded in Italy, Switzerland and Verdun (A.D. 560, Gregory of Tours), while according to Newman the first leper house in England was founded in Nottingham in A.D. 625. Creighton, however, gives A.D. 936 as the date of the earliest leper hospitals, founded by King Athelstane at York (St. Peter's and St. Leonard's). Legislative enactments regarding the marriage of lepers were also made by Rothar, King of the Lombards, in the seventh century, and by Pepin and Charlemagne in the eighth century. The invasion of Spain and France by the Saracens may have helped to increase the disease in these countries in the seventh and eighth centuries.

The first leper hospital in Ireland was founded in the ninth century, and the first leper hospital in Scotland in the tenth century, while in the same century we hear of an edict, similar to that of Pepin, being promulgated in Wales. In the tenth and eleventh centuries leprosy increased in all the countries we have mentioned. It did so in England particularly after the Norman invasion, when several leper houses were founded. In the eleventh century the first leper house in Spain was founded. At the end of the twelfth century it first became prevalent in Denmark, whence it was carried to Iceland. From Scotland the disease appears to have been carried to Norway (a leper house was founded at Bergen in 1266), while from Norway it was carried by the Vikings to the Faroës, to the Shetlands and to Greenland.

During the twelfth century leprosy was becoming so alarming that it became the practice to persecute lepers in Europe, and at this time the famous writ *"de Leproso Amovendo"* was promulgated in England. In the thirteenth century, at the time of the return of the Crusaders, leprosy reached its zenith in England and in south-western Europe, while it was increasing in Scotland, Holland, Denmark, Sweden, Norway, the Baltic, the Black Sea and the Crimea. At this time there were 2,000 leper houses in France alone, and numerous leper houses were being founded in England.

The fourteenth century marks the commencement of the decline of leprosy in England and south-western Europe, although it was still on the increase in the north-western countries of Europe. In Scotland, leper houses were being founded at Kingcase, Glasgow and Govan, while at this time Robert the Bruce is said to have died of leprosy. By the fifteenth century leprosy had become uncommon in Italy. In Switzerland, however, numerous leper houses were being founded.

While leprosy appeared to be on the wane in Europe, two significant events occurred, namely, the increase of syphilis to epidemic proportions and the discovery of America, to which leprosy was destined to spread.

During the sixteenth century, leprosy disappeared almost entirely from Italy, save in a few isolated areas, and during the same period it underwent a considerable decrease in Spain and France. In Germany and the Netherlands leper houses were still being founded; but in England leprosy had by now become a rare disease, save in Cornwall. In Scotland and in north-west Europe it was still prevalent, although in Denmark it became extinct at this time.

By the seventeenth century there were so few lepers in France that leper houses were abolished by Louis XIV. Leprosy was still common, however, in Lorraine, and certain endemic foci persisted in Auvergne and Belle-Isle down to the end of the nineteenth century. In the Netherlands it died out towards the end of the seventeenth century. Meanwhile it was decreasing all over the British Isles, but remained prevalent in the Faroës, Iceland, Shetland and St. Kilda.

In the eighteenth century leprosy became practically extinct in the British Isles, the last leper in Scotland, a Shetlander, having been admitted to the Royal Infirmary, Edinburgh, in 1798. In France it had almost died out save in Auvergne, and in that country a few cases may be found to this day in the Maritime Alps.

In the nineteenth century leprosy was not only widespread, but actually increasing in Norway; it was not quite extinct in Germany; while there were many lepers left in Iceland, Sweden, the Baltic, Spain, Portugal and Italy.

During the period that leprosy was decreasing in Europe it was carried to the West Indies and America by the Spanish and Portuguese conquerors and colonists. There seems to be no good evidence that leprosy existed on the American continent in pre-Columbian times. Cortes is said to have founded a leprosarium soon after his arrival in Mexico, but this is doubtful, and in any case the hospital, if it existed, may have been for the care of sufferers from syphilis and other diseases. The native races to this day have remained free from leprosy, save where they have come into close intercourse with immigrants. The buccaneers also appear to have been responsible for the spread of leprosy from West Africa to the West Indies and America, the disease, according to Ehlers, having been common among the negro slaves. Leprosy appeared among the white population in the south of the United States of America first in Louisiana in 1758, having probably been imported from the West Indies. It was carried to New Brunswick, Canada, in 1758, by a Frenchwoman from St. Malo, while some Norwegian lepers carried the disease to Minnesota, where it has died out. Indians and Chinese may have been responsible for carrying it to the western seaboard of Canada and the United States of America and also to the West Indies. Leprosy has spread throughout Africa and the Sudan chiefly through the opening up of trade routes and the transport of slaves.

In the nineteenth century a remarkable outbreak of leprosy occurred in the islands of the Pacific, infection having been carried thither by the Chinese. First Hawaii was infected in 1848, and later New Caledonia in 1865. From New Caledonia the disease spread in 1878 to the Loyalty Islands and to the Marquesas Islands. The Chinese, according to Jeanselme, have also been the means of carrying the disease to Indo-China, Siam, the Straits Settlements, Java, Burma, the Philippines and other East Indian islands.

We are indebted to the studies of Ashburton Thompson (1897) and Cook (1927) for an account of the history of leprosy in Australia. It appears from the studies of these writers that leprosy was unknown among the aborigines, but that it was introduced by Chinese immigrants during and after the sixth decade of the nineteenth century and by Polynesians a decade later. There is no evidence of its having been imported by any of the whites. In the northern parts of Queensland the advent of the

Chinese and Polynesians led to the infection of the aborigines, who in turn communicated it to the whites through intimate contact, native women having been the chief intermediaries. In New South Wales leprosy appeared in the pioneering communities after the immigration of infected aliens. Owing to the early disappearance of the aborigines from that State, it was communicated from white to white and became a domestic disease. In Victoria, the disease never became widespread, for although it was introduced by Chinese, there was no aboriginal population on the goldfields to act as intermediary (Cook, 1927).

Regulations for the Dealing with Lepers.

The treatment which the leper has received at the hands of his fellow men shows a curious inconsistency. On the one hand he has been regarded as unclean, an outcast to be shunned; on the other hand he has been the object of the most ostentatious solicitude, and surrounded by a special halo of sanctity. This arises largely out of the biblical associations of leprosy and the religious sentiment with regard to lepers. In the Old Testament the harsher sentiment predominates. The orders of the priests as given in Leviticus xiii, 45, 46, were as follows:

> And the leper in whom the plague is, his clothes shall be rent, and his head bare, and he shall put a covering upon his upper lip, and shall cry, Unclean, unclean. All the days wherein the plague shall be in him he shall be defiled; he is unclean: he shall dwell alone; without the camp shall his habitation be.

The harsh edicts and regulations relating to lepers in the Middle Ages all echo and seek to amplify the above. The leper had to wear a special grey and russet gown, to give special warning of his approach by means of a bell or clapper, and to cry "Unclean, unclean". He was also often compelled to live outside the town. For example, the measures ordained by the burgers of Berwick-on-Tweed were as follows:

> No leper shall come within the gate of the borough; and if one gets in by chance, the sergeant shall put him out at once. If one wilfully force his way in, his clothes shall be taken off him and burnt and he shall be turned out naked. For we have already taken care that a proper place for lepers shall be kept up outside the town and that alms shall be there given to them.

On the other hand, the attitude of Jesus toward the leper in the New Testament was one of pity and help, and this sentiment likewise finds expression in the Middle Ages. The picture of the leper became associated with that of Lazarus in the parable in Saint Luke's gospel, the man full of sores who was laid at the rich man's gate. By a curious association of ideas the image of this Lazarus, a purely allegorical figure, became fused with that of a real man, namely, the Lazarus of Saint John's gospel, the brother of Martha and Mary, the man whom Jesus loved and whom he raised from the dead, but who we have no reason to believe was a leper. Nevertheless, it was this Lazarus of Bethany who became the patron saint of lepers. It followed from this composite picture of the two Lazaruses that the leper was at once one of "Christ's poor" and "beloved of God". The care of the leper came, therefore, to be regarded as a religious duty, and this not infrequently passed into the realm of personal devotion. For example, of Walter de Lacy the chronicler of Battle Abbey writes:

> He especially compassioned the forlorn condition of those afflicted with leprosy and elephantiasis, whom he was so far from shunning, that he frequently waited upon them in person, washing their hands and feet and with the utmost cordiality imprinting upon them the soothing kisses of love and pity.

King David of Scotland related to the Abbot of Rivaulx the following story regarding his sister, Queen Matilda, wife of Henry I:

> When he was serving as a youth at the English court, one evening he was with his companions in his lodgings when the Queen called him into her chamber. He found the place full of lepers and the Queen standing in the midst with her robe laid aside and a towel girt about her. Having filled a basin with water she proceeded to wash the feet of the lepers and to wipe them with the towel, and then, taking them in both her hands she kissed them with devotion. To whom her brother—"What doest thou, my lady? Certes, if the King were to know this, never would he dream to kiss with his lips that mouth of thine polluted with the soil of lepers' feet!" But she answered with a smile: "Who does not know that the feet of an eternal king are to be preferred to the lips of a mortal king? See then, dearest brother, wherefore I have called thee, that thou mayest learn by my example to do so also. Take the basin and do what thou hast seen me do!" At this, said David, I was sore afraid and answered that I could on no account endure it. For as yet I did not know the Lord, nor had His spirit been revealed to me. And as she proceeded with her task, I laughed—*mea culpa*—and returned to my comrades.

This sentimental attitude towards lepers also found expression in the formation of an order of knights under the banner of Saint Lazarus. It was in fact the most sentimental of all the orders of chivalry. It probably branched off from the Knights Templars or main body of Hospitallers, but according to some the order goes back to A.D. 366, long before the Crusades. They had their headquarters in Jerusalem, the grand master and the knights there being "leprosi". Creighton (1891) says that we should interpret the term "leprosi", as applied to them, in a liberal sense, and that we should be doing them no injustice if we took them to have been Crusaders so badly hit by their vices or other misfortunes as to be marked off as a separate order. It is not known what services they actually performed for lepers, but they were disliked by the clergy, who said of them *"Rapiunt ut distribuant"*, which implies that they were not too scrupulous in their dealings.

Undoubtedly the most important aspect of this religious movement in favour of lepers was the formation of leper hospitals (Simpson, 1841 and 1872; Creighton, 1891; Clay, 1909; Mercier, 1915; Newman, 1858; Virchow, 1860-1861; Ehlers, 1899). These were for the most part dependencies of the abbeys, and we note a very great disproportion between the number of patients and the clerical staff. Many of these institutions, though in the main intended for lepers, were used as hospitals for the

sick poor generally. It would probably be a mistake to suppose that the earlier hospitals, at any rate, were founded as a prophylactic measure, with the idea of segregating sufferers from an infectious disease. It is true that Rhazes and Avicenna had declared leprosy to be contagious, but it is doubtful whether professional medical opinion played any important part in promoting measures for the treatment and segregation of lepers so early as the twelfth century. Among the clergy and the lay public the idea of there being any danger in coming into contact with lepers does not seem to have entered into the question at this time. It was rather the biblical notions regarding leprosy, the fact that the leper was a loathsome object, unclean and unfit to associate with men, which led to his being sheltered in these institutions. The segregation of lepers seems in the first place to have been a sort of benevolent ostracism, and not only lepers, but sufferers from many other loathsome diseases seem to have been included in the category of "leprosi".

The popular and clerical opinion regarding the contagiousness of leprosy seems, however, to have developed strongly towards the close of the twelfth century. The canon *"de Leprosis"* (Lateran Council, Rome, 1179) declares emphatically that lepers cannot dwell with healthy men. We also read how the prior of Taunton separates a monk from his brethren "in fear of the danger of this illness"; while a Durham chronicler mentions an infirmary for those "stricken with the contagion of leprosy". The English civil law set its seal upon the theory of infection by the writ *"de Leproso Amovendo"* (A.D. 1100), authorizing the expulsion of lepers on account of the manifest peril of contagion. Long before this, however, the expulsion of lepers had become a custom, and it was largely a matter for local legislation.

The duty of reporting and examining cases fell to the clergy, to doctors and to civil officers, or to a jury of "discreet men". In ordinary cases the leper would show himself to the parish priest, as the only scholar. Although leprosy became a penal offence, only laymen could be cited and dealt with by the jury, mayor or feudal lord. The clergy had to answer to their bishop. No doubt many mistaken diagnoses were made, but it is also probable that when the disease was so common responsible persons would have become familiar with its important features. There was also justice for anyone falsely accused of being a leper; when mistakes had been made, the civil authorities were sometimes severely rebuked and ordered to give redress. The law evidently had no power to touch a leper unless he made himself a source of public danger. No one interfered with him so long as he remained in a quiet hiding place, quitting it, perhaps, only at night. Individuals sheltered by the affection or self-interest of relatives might escape the law. But once adjudged a leper, or coming under the ban of the law, the poor victim became an outcast. He lost his civil rights, he could neither inherit property nor bequeath it; his wife could divorce him and marry another man; he was expelled from the community and segregated. He became civilly dead. The removal of a leper became a matter of clerical ritual, one sufficiently calculated to impress the sufferer, as may be gathered from the following description of the office at the seclusion of a leper (Clay, 1909, appendix):

First of all the sick man or the leper, clad in a cloak and in his usual dress, being in his house, ought to have notice of the coming of the priest, who is on his way to the house to lead him to the church, and must in that guise wait for him. For the priest, vested in surplice and stole, with the cross going before, makes his way to the sick man's house and addresses him with comforting words, pointing out and proving that if he bless and praise God, and bear his sickness patiently, he may have a sure and certain hope that though he be sick in body he may be whole in soul and may reach the home of everlasting welfare. And here, with other words suitable to the occasion, let the priest lead the leper to the church, when he has sprinkled him with holy water, the cross going before, the priest following, and last of all the sick man. Within the church, let a black cloth, if it could be had, be set upon two trestles, at some distance apart, before the altar, and let the sick man take his place on bended knees beneath it between the trestles, after the manner of a dead man, although by the grace of God he yet lives in body and spirit, and in this position let him devoutly hear mass. When this is finished and he has been sprinkled with holy water, he must be led with the cross through the presbytery to a spot where a pause must be made. When a spot is reached the priest shall counsel him out of Holy Scripture, saying: "Remember thine end and thou shalt never do amiss. Whence Augustine says: 'He readily esteems all things lightly, who ever bears in mind that he will die'." The priest then with a spade casts earth on each of his feet, saying: "Be thou dead to the world, but alive again with God." And he comforts him and strengthens him to endure with the words of Isaiah: "Truly he hath borne our griefs and carried our sorrows, yet we did esteem him as a leper, smitten of God and afflicted" (Isaiah, liii, 4).[1] Let him say also: "If in weakness of body by means of suffering thou art made like unto Christ, thou mayest surely hope that thou wilt arise in spirit with God. May the Most High grant this to thee, remembering thee among the faithful ones in the book of life." It is to be noted that the priest must lead him to the church, from the church to his house, as a dead man, chanting the *Responsorium Libera me Domine* in such wise that the sick man is covered with a black cloth. When leaving the church after mass, the priest ought to stand at the door to sprinkle him with holy water, and he ought to commend him to the care of the people. Beforehand, the sick man ought to make his confession in the church, and never again. When he has come into the open fields he ends by imposing prohibitions upon him in the following manner: "I forbid you ever to enter church, or to go into a market, or mill or bake house, or visit any assemblies of people. Also I forbid you ever to wash your hands or any of your belongings in spring or stream of water of any kind, and if you are thirsty you must drink water from your cup or some other vessel. Also I forbid you ever henceforth to go out without your leper's dress, that you may be recognized by others, and you must not go outside your house unshod. Also I forbid you, wherever you may be, to touch anything which you may wish to buy, otherwise than with a rod or staff to show what you want. Also I forbid you ever henceforth to enter taverns or other houses if you wish to buy wine; and take care even that what they give you they put into your cup. Also I forbid you to have intercourse with any woman except with your own wife. Also I command you when you are on a

[1] The biblical quotations in this office are not from the Authorized Version.

journey not to return an answer to anyone who questions you, till you have gone off the road to leeward, so that he may take no harm from you; and that you never go through a narrow lane lest you should meet someone. Also I charge you if need require you to pass over some toll way (*pedagium*), through rough ground or elsewhere that you touch no posts or things whereby you cross till you have first put on your gloves. Also I forbid you to touch infants or young folk, whosoever they may be, or to give them or to others any of your possessions. Also I forbid you henceforth to eat or drink in any company except that of lepers. And know that when you die, you will be buried in your own house, unless it be, by permission obtained beforehand, in the church." And note that before he enters his house he ought to have a coat and shoes of fur, his own plain shoes and his signal the clapper, a hood and a cloak, a pair of sheets, a cup, a funnel, a girdle, a small knife, and a plate. His house ought to be small, with a well, a couch furnished with coverlets, a pillow, a chest, a table, a seat, a candlestick, a shovel, a pot and other useful articles. When all is complete the priest must point out to him the ten rules which he has made for him and let him live on earth in peace with his neighbour. Next must be pointed out to him the ten commandments of God, that he may live in heaven with the saints, and the priest repeats them to him in the presence of the people. And let the priest also point out to him that every day each faithful Christian is bound to say devoutly the *Paternoster*, *Ave Maria*, *Credo in Deum*, and *Credo in Spiritus*, and to protect himself with the sign of the cross, saying often "*benedicite*". When the priest leaves him, he says: "Worship God and give thanks to God. Have patience and the Lord will be with thee. Amen."

While in the leper house the leper was under strict religious discipline, but he was allowed a certain amount of freedom in his movements. He could go out and beg, he might visit his wife, provided that he did not stay away for more than a limited period—three hours according to Cabanès. He might in some places, attend divine service, but he had to look through a special window or grating, and use a separate receptacle for holy water. Nevertheless, if he attempted to abscond, he was severely dealt with. A gallows outside the hospital acted as a warning. The leper house was in no sense intended for treatment, but merely as a charitable retreat, the disease being regarded as incurable. Indeed the case is recorded of a certain Christine Livingstone, tried for witchcraft, of whom the general accusation was that "she affirmit that she culd hail [cure] leprosie, quhilp the maist expert men in medicine are not abil to do". (Pitcairn's "Criminal Trials in Scotland", Volume II.)

The practice of isolating lepers in villages has prevailed in many parts of the world, especially in China, Africa and Oceania, but segregation has been imperfect. In Scotland we have evidence of a leper village, which was called Liberton or Leper Town, having existed near Edinburgh.

The compulsory segregation of lepers in modern times has been extensively practised, but the stringency of the regulations and the continuity with which they have been put into effect have varied from time to time with the fluctuation of opinion regarding the contagiousness of leprosy. It was relaxed in many places when the theory of the hereditary transmission of leprosy, advocated by Danielssen and Boeck, held the field, and again after the publication of the report of the Royal College of Physicians of London in 1862, which declared against the contagiousness of leprosy. In each case, whenever segregation fell into abeyance, an increase of leprosy is said to have followed (Rogers and Muir, 1925). The most remarkable results of segregation are claimed in Norway, where the isolation of lepers was begun in 1856, the numbers having now been reduced almost to vanishing point. Home supervision, combined with the segregation of the more infective cases, has also been successfully practised in Norway, Madagascar, Australia, and elsewhere.

Of the more barbaric methods of dealing with lepers it is hardly necessary to speak. In England especially lepers were not hardly dealt with, but in France they were frequently persecuted, Philip V having burnt lepers on the pretext that they poisoned the wells. Philip's methods have been paralleled in recent times by native practices in Sumatra, while barbarous methods have frequently been in vogue among native tribes in various parts of Africa.

The Disappearance of Leprosy.

We now come to the interesting question of the cause of the decline or complete disappearance of leprosy in countries where it was formerly endemic. We have seen how leprosy steadily spread over Europe and how, broadly speaking, it declined first in those areas first attacked and persisted longest at the periphery. *Prima facie*, this looks as if some change had followed in the wake of the epidemic, making the conditions unfavourable for its continuance. What was it that had changed? Was it the lepra bacillus itself, was it the human soil, or was it the environmental conditions?

With regard to the first of these, it is known that diseases alter their character, that organisms may change their virulence, either as the result of mutations, or as the result of the changed conditions of their growth; for example, the virulence may be heightened by passage through susceptible stock. If a fall in virulence was due to the stock having become less susceptible, then it would be to the soil rather than to the seed that we should have to look for the ultimate explanation of the change. In any case, there is no reason to believe that the leprosy bacillus has lost its virulence at the present day, in view of its ravages among susceptible races such as the Polynesians and the negroes of central Africa.

We come, therefore, to the second possibility, namely, that there has been a change in the nature of the soil. Under this heading we have to consider at least three ways in which the soil may become altered: first, that immunity acquired in one generation may be transmitted by inheritance to succeeding generations; secondly, that susceptible individuals may be weeded out, leaving behind them more resistant stock, the general level of immunity in the race being thus raised; and thirdly, that long

endemicity may produce a higher proportion of individuals immunized by small doses of the organism.

The first of these possibilities may be dismissed as being entirely unsupported by evidence and in conflict with the general law that acquired somatic characters are not transmissible. The second theory, namely, that there has been a change in racial immunity brought about by elimination of the more susceptible stock, requires more serious consideration than it has hitherto received, and it has been strongly advocated by Molesworth (1926, 1932). By referring the disappearance of leprosy to a single cause it has the advantage of economy of hypothesis, and it is by far the most comprehensive explanation which has been offered. It is, however, one of those tantalizing hypotheses which are as difficult to disprove as they are to prove, because of the difficulty of getting the necessary data to settle the question. There can be no doubt that considerable differences in natural immunity exist between different species and between individuals of the same species, and it has been shown experimentally that such inherited susceptibility may be transmitted according to simple Mendelian ratios. But while it is possible by careful breeding to produce relatively immune stock, it is equally certain that in the case of some species natural immunity appears to be accidental, there being no reason to believe that it has arisen specifically as a result of natural selection in relation to a particular organism. When, therefore, differences in susceptibility to leprosy are noted among the different races of mankind, the real question to be determined is whether these differences, if they truly exist, have or have not been present *ab initio*, or whether they have resulted from the operation of natural selection *vis-à-vis Bacillus leprœ*. The assumption that races recently attacked suffer most severely because natural selection has not previously eliminated the susceptible members of the stock, is a begging of the question. If we knew exactly how the susceptibility of the inhabitants of Great Britain before or during the height of the epidemic compared with that of their descendants of the present day when placed under similar conditions; if we knew how the Polynesians would behave with respect to leprosy infection ten or twelve generations after the beginning of the outbreak and under uniform conditions, supposing always that the race remained pure, we might have some data to go on. It may also be doubted whether it may be accepted as a general law that races amongst which the disease has long been present have always developed natural immunity. Leprosy endured in countries like Denmark and Britain for only a thousand years or less, but it is reputed to have been endemic in Egypt for at least twice that time, while the Sudan and central Africa, which are believed by some (on slender grounds, it is true) to have been the cradle of leprosy, still rank among the countries showing the highest case incidence. It is also an undoubted fact that Europeans of the present day may become attacked when exposed to infection. It is possible, however, that this may be due to the passage of the organism through some highly susceptible stock, whereby its virulence would be raised, and this may account for the occurrence of leprosy among whites in Australia, Polynesia and Africa. It is also contended that in these countries and in India, the effects of a tropical climate upon Europeans may diminish the resistance to infection. On the other hand, as recently as last century (1880) an outbreak of leprosy occurred in Memel, where it was introduced by five infected Russian servant girls; again, the outbreak in New Brunswick (1815) had as its starting-point a French immigrant from St. Malo (Rogers and Muir). In neither case could passage through a highly susceptible race nor the effects of a tropical climate be adduced as favouring circumstances. Further, Cook points out that in certain districts of New South Wales and Queensland (Lismore and Rockhampton) exceptionally virulent outbreaks of leprosy occurred among Europeans, quite apart from intermediate transmission through the aborigines. On the other hand, cases of leprosy have from time to time been introduced into France and Great Britain from abroad without spreading infection; this may have been due to the cases having been of a mild and feebly infective maculoanæsthetic type or to the absence of the external conditions necessary for the spread of contagion, of which we at present know very little.

Perhaps the greatest difficulty in accepting the racial immunity theory is that we should have to assume the existence of a very high rate of elimination or of exclusion from the possibility of reproduction of individuals in an infected community if the proportion of non-susceptible to susceptible persons was to be raised to any significant extent. In the case of highly infectious diseases, of which the infecting organism becomes practically ubiquitous and of which the mortality is very high, natural selection would obviously be of importance; and this probably accounts for the difference in susceptibility to plague between Madras rats and Bombay rats. With the high leprosy case incidence —200 per thousand—met with in central Africa and Polynesia, there would appear to be some likelihood that after a number of generations a general elevation of racial immunity would occur. We have to remember, however, that in England the height of the epidemic (when the proportion of susceptible individuals must have been high) occurred in the thirteeenth century, while two or three centuries later, or approximately eight to twelve generations, it had practically disappeared. To raise the general level of racial immunity to any significant extent in a small number of generations would require a very high elimination rate, but of this we have insufficient evidence. Creighton computes that the case incidence was at no time greater than about two or three per thousand, and he goes so far as to say that in mediæval England the village leper may have been about as common as the village fool. If this is correct, natural selection must have been a negligible factor. It is not possible, however, to arrive at any

accurate estimate of the prevalence of leprosy in the Middle Ages. It may be contended on the one hand that many people were regarded as lepers who were in reality suffering from other diseases, such as syphilis; on the other hand, it is probable that cases of genuine leprosy were often overlooked, owing to the difficulties in diagnosis. In any event, it seems extremely doubtful whether Creighton's estimate of the incidence of leprosy in the Middle Ages can be accepted as even approximately correct. In view of the definite information we now possess regarding the character and incidence of leprosy as observed over the last one hundred and fifty years, when the disease has been sown upon virgin soil and the similarity of the incidence among peoples widely separated geographically and racially has been noted, Creighton's statement must be regarded with scepticism.

The question as to whether the development of racial immunity through natural selection is adequate to explain the decline of leprosy reduces itself to the following: what rate of incidence of the disease and what rate of elimination of susceptible individuals would be required to cause leprosy to disappear in the eight to twelve generations between the height of the epidemic and the virtual disappearance of the disease? Would the theoretical requirement correspond to the known facts? This is a question which requires to be attacked mathematically on the lines of J. B. S. Haldane's treatment of the general problem of natural selection. The difficulty in dealing with such a problem lies in the extreme complexity of the factors involved and in the inadequacy of statistical data.

We have now to consider the third way in which immunity may be produced. When a disease attacks virgin soil, it is commonly characterized by a short incubation period and a high mortality, while in endemic areas it is marked by a high morbidity but a low mortality. If we take tuberculosis as a parallel, the same differences are observed between town and country as between endemic and non-endemic areas. This has been explained by the supposition that in endemic areas a large number of individuals become immunized by small doses of the bacillus so that the disease occurs only in a mild form. In the case of diseases of low infectivity, the comparatively healthy carrier, shedding few bacilli, may become more a source of immunity than of danger, while the reverse is the case with a highly infective organism. Leprosy would fall into the former category, but unfortunately we know very little about this aspect of the epidemiology of the disease. Kitasato, in Japan, examined the nasal mucus of sixty-eight healthy persons in the families of lepers who were living with them, and found very numerous bacilli in eight of them. It is possible that such carriers may, by disseminating small doses of bacilli, produce relative immunity in contacts, so that in an endemic area the number of such individuals would become relatively great, and eventually the milder forms of the disease, such as the maculo-anæsthetic type, would begin to predominate. These being the least infectious, the spread of infection would become less rapid and the epidemic would limit itself. It would be unprofitable, however, to speculate further along these lines; what we require is further investigation.

The next theory we have to deal with ascribes the disappearance of leprosy to changes in the environment, and especially to changes in the social conditions and habits of the people. Generally speaking, leprosy was at its height in Europe when the social conditions were at their worst, and declined at the close of the Middle Ages with the Renaissance and the rise of city States in Italy, and with the spread of culture together with increase of trade and prosperity throughout Europe. In the case of a disease of very low infectivity like leprosy, a very slight or subtle change in environmental conditions might make a big difference. But the difficulty arises when we attempt to discover the relevant factors. First, the dietetic factor has to be considered. Famines were common throughout the Middle Ages, and it seems probable that general inanition would lower the resistance to infection; but famines continued to occur even during the period of decline, and it does not seem necessary to assume that such marked privations are essential to the spread of leprosy. Was there, then, some more specific dietetic factor? Jonathan Hutchinson (1890) thought that the eating of fish, especially rotten fish, was responsible for leprosy; he pointed out that leprosy increased when fast days became more numerous through ecclesiastical influences and when fish was the main source of animal food, while it declined when the practice of fish-eating passed out of vogue in the fourteenth century. The doctrine that leprosy is caused by the consumption of fish is, of course, an exploded one, since leprosy occurs among non-fish-eating peoples; but it is necessary to review the fish theory in the light of modern work upon accessory food factors. Fish are singularly lacking in these, especially white fish—not so much fat fish like salmon—which were largely consumed by the peasantry. If vitamin deficiency was responsible, to which of the specific vitamins could the susceptibility to leprosy have been related? The diet of people in the Middle Ages must have been poor in vitamins *A* and *B*, as few green vegetables were grown; but it is important to note that leprosy had declined and almost disappeared from England before the introduction of market gardening. Catherine of Aragon in the sixteenth century had to send for a Dutch gardener to grow salads for her.

The story of leprosy is quite different from that of scurvy. This disease was very common in mediæval England, because the cattle had to be killed in the autumn and salted down, and there was a lack of both fresh meat and fresh vegetables during the winter. But scurvy did not disappear from England until after the sixteenth century, when green vegetables and root crops were introduced from Holland, and cattle could be fed on turnips during the winter, making fresh meat available. Long before this, however, leprosy had been on the wane. Vitamin *A* deficiency is common amongst peoples living on an almost exclusively

ILLUSTRATIONS TO THE ARTICLE BY DR. WILFRED EVANS.

FIGURE I.
Photomicrograph of section of the bundle of His in a sheep's heart. (×70.)

FIGURE II.
Photomicrograph of section of the bundle of His in a sheep's heart. (×245.)

FIGURE III.
Photomicrograph of section of the bundle of His in a bullock's heart. (×70.)

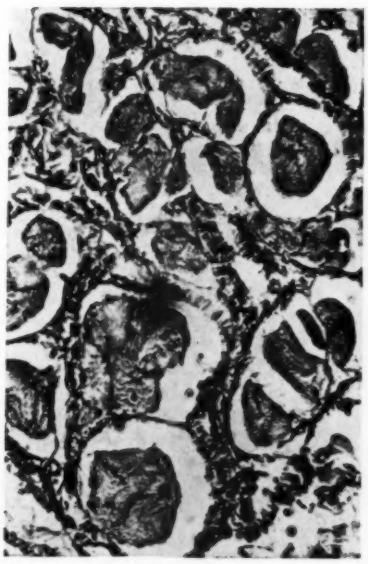

FIGURE IV.
Photomicrograph of section of the bundle of His in a bullock's heart. (×245.)

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Figure V.
Photomicrograph of section of the bundle of His in a human heart. (×70.)

Figure VI.
Photomicrograph of section of the bundle of His in a human heart. (×70.)

Figure VII.
Photomicrograph of section of the bundle of His in a human heart. (×245.)

Figure VIII.
Photomicrograph of section of the bundle of His in a human heart. (×245.)

ILLUSTRATIONS TO THE ARTICLE BY DR. WILFRED EVANS.

FIGURE IX.
Photomicrograph of section of the bundle of His in a human heart. (×245.)

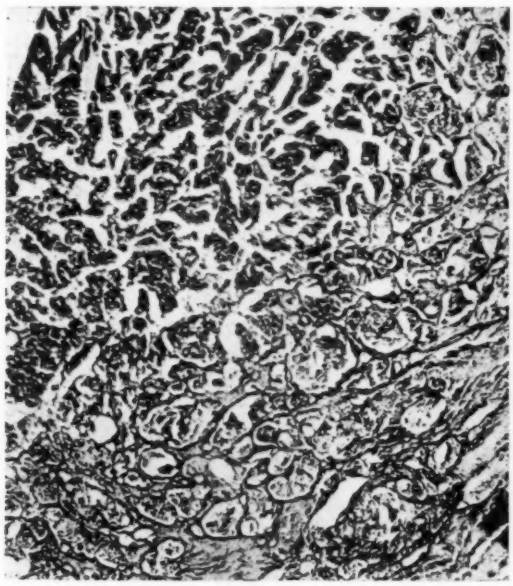

FIGURE X.
Photomicrograph of section of the bundle of His in a human heart. (×245.)

ILLUSTRATION TO THE ARTICLE BY DR. H. LEIGHTON KESTEVEN.

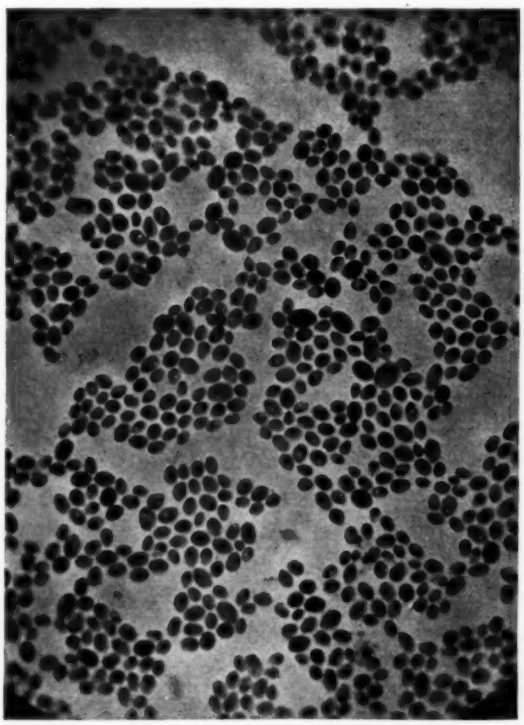

FIGURE II.
Microscopic appearances of *Eutorula excorians* Kesteven. (×800.)

ILLUSTRATION TO THE ARTICLE BY DR. A. WALKER-SMITH.

FIGURE I.
Skiagram of lower part of urinary tract, showing calculi in bladder and urethra.

ILLUSTRATIONS TO THE ARTICLE BY DR. M. P. SUSMAN.

FIGURE II.
Postero-anterior view, taken immediately after the breaking of the needle, which is in the thoracic wall.

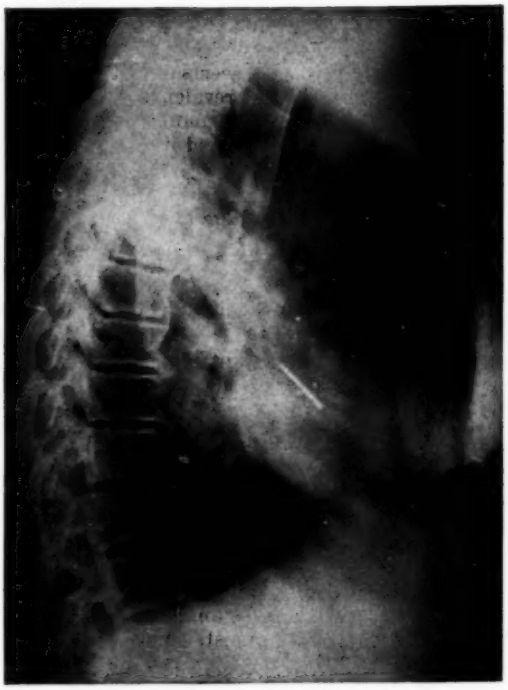

FIGURE III.
Lateral view, taken immediately after the breaking of the needle; it is in the thoracic wall.

FIGURE IV.
Postero-anterior view, taken after the first attempt to remove the needle; it is now in the pleural cavity.

FIGURE V.
Lateral view, taken after the first attempt to remove the needle; it is now in the pleural cavity.

fish diet, as in parts of the Malay Peninsula, Norway and Labrador; but in the Middle Ages the consumption of rye bread and lentils may have helped to protect against this form of deficiency, although in England rye was scantily cultivated.

Another possibility which seems to fit in somewhat better with the conditions prevalent in the Middle Ages and amongst leprous communities in central Africa, India, Malay, China and Polynesia at the present day, is that a deficiency of protein of mammalian origin may have rendered the individuals susceptible. During the fourteenth century people began to eat less fish; and the change from tillage to pasture, and the growth of the wool industry and the rearing of more sheep for wool and mutton led to the consumption of more mammalian flesh in the form of mutton and beef. Pork was also an important source of mammalian protein. We have no experimental evidence regarding the effect of lack of mammalian protein or even of general protein deficiency upon the susceptibility to disease; but the whole question of dietetic deficiency and its relationship to susceptibility to leprosy requires further experimental investigation.

Another factor to be reckoned with is that of propinquity. Leprosy requires very close and prolonged contact in order that it may be communicated from one individual to another, bed infections being amongst the commonest. In this connexion it is interesting to note that in the fourteenth century there was an increase in privacy among the well-to-do; houses had more rooms, and instead of several people sleeping in one bed or in one room, they began to have separate rooms. The practice of wearing night-dresses was also introduced at this time. Hansen relates how leprosy disappeared from amongst a community of Norwegians who had settled in Minnesota, and who adopted the habit of sleeping in separate rooms instead of several in one bed, as had been the custom in Norway. While the improved conditions referred to may have prevailed amongst the wealthy classes in the fourteenth century, this was not the case with regard to the lower orders. Real and general improvement in housing conditions did not appear until Elizabethan times.

At this point it is interesting to refer to the effects of plague, and especially to the pandemic known as the "Black Death", which swept Europe, simultaneously with the disappearance of leprosy. The Black Death may have acted partly by diminishing propinquity through thinning the population, which is said to have been reduced by half, and also by killing off the lepers, already weakened by disease, unable to help themselves, and crowded in possibly flea-infested leper houses and hovels. There is a theory that leprosy is communicated from the rat to man by means of the flea, and it is supposed by some that the Black Death, owing to its virulence, may have killed off the rats, thereby removing the source of infection. There is, however, no evidence that rat leprosy can be communicated to man, and leprosy occurs in places where there are no rats; but whether leprosy is spread by an insect vector from man to man is an important question which requires further elucidation. Until we have information on this point it is impossible to assess the importance of the various social factors under review.

The Black Death also helped to precipitate certain changes in rural conditions which may perhaps be relevant. About this time the system of enclosures was introduced, and this led to the disappearance of the village community, in which land was held in common while the labourer received payment in kind from the lord of the manor. The labourer now became more independent, and received wages in money in return for his services. Simultaneously the rise of the weaving industry caused many to drift into towns, such as Norwich. In the country the conversion of land from tillage to pasture and the inability of the lords of the manor to pay wages led to an increase in vagabondage. How did the leper fare under these conditions? At this epoch we have seen that there was a strong sentiment against lepers, legislation against them having become harsher, and the plague may have helped to make people familiar with the idea of contagion. When the leper was segregated it might now mean the removal of a wage-earner, and the rest of the community and the lord of the manor would be under no obligation to keep his family. Consequently the leper and his family might be worsted in the struggle for life, and even die of starvation. It is in conjunction with such conditions that we have to consider the influence of segregation. It is believed by many that the seclusion of lepers had little to do with the disappearance of leprosy because it was inefficiently practised, lepers being even allowed to go out to beg. It is also held by some that since segregation has been shown to have little influence upon the spread of a highly infective disease like scarlet fever, it would be still more useless in the case of one of a low infectivity like leprosy. This, however, by no means follows. In the case of a disease of high infectivity, carriers, as already pointed out, are a source of danger as well as of immunity, massive doses of infection being unnecessary to communicate the disease; but in the case of a disease of low infectivity, carriers would not be such a danger and they would be more likely to produce immunity.

We learn from the writings of Gordon, John of Gaddesden and others, that it was when the form and figure of the face became altered that segregation was considered necessary. This means that it was more especially victims of nodular leprosy of the face who were selected for segregation. In the light of modern work, we know that these are precisely the more infective ones, discharging as they do large numbers of bacilli from the nose and thereby tending to bring about massive infection. Sufferers from the maculo-anæsthetic variety, even if they were not so rigorously segregated, would prove less dangerous to the community.

When once a man was adjudged a leper and segregated, not he alone, but all his family would suffer. In some places, as in parts of France, not only lepers, but those living with them were required to give warning to others by means of a special sign, such as a goose's foot imprinted on their garments. The persecution and ostracism, not only of the leper, but of those likely to have been infected by him, the poverty or even starvation which might overtake the family of the bread-winner, would lead, as Liveing says, to the extermination of the race of lepers. This would not necessarily occur, however, by elimination of the susceptible members of the stock, but by stamping out of the reservoirs of infection, by extermination of those individuals who were infected or coming within the circle of those most likely to have acquired infection. At the present day the lot of the leper, although hard indeed, is much happier than that of his predecessors in former generations. It is hoped that the proper supervision of patients, judicious segregation of the more infective ones, together with the development of efficient methods of treatment, may ultimately lead to the eradication of the disease.

Acknowledgements.

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THE BUNDLE OF HIS.

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IN recent years much interesting work has been published on that very important part of the heart, the bundle of His. Most of this research has been performed on animals, and in the ox its arborization has been demonstrated by injection of its sheath with indian ink.

Animal experiments have also shown that the electrocardiographic tracing which we knew as due to right bundle block is in effect due to left bundle block; but we have less accurate knowledge of the bundle and its branches in the human heart. As yet it has been impossible to confirm animal experiments by human pathology. There is such difficulty in demonstrating the bundle of His in the human heart that some observers have even doubted its existence—a doubt which prompted me to investigate this field of work.

With Dr. A. C. Telfer, I readily found and dissected the bundle of His and its branches in the heart of the bullock and sheep. In Figures I to IV the large pale Purkinje fibres can be seen. In the human heart the task was much more difficult; Purkinje fibres rapidly degenerate after death, and only in very fresh specimens could satisfactory results be obtained. Added to this, in no specimen was I able to see the bundle with the naked eye. Blocks were taken from the lower part of the right auricle and the upper part of the interventricular septum on both sides; yet so small is the bundle that examination of serial sections failed, in some cases, to reveal any Purkinje cells. A third difficulty was that we could discover no satisfactory method of differential staining. After working with Van Giesen's and Sabin's modification of Mallory's stain, we found that eosin-hæmatoxylin gave the best results.

We traced the main bundle of His from the coronary sinus to the *pars membranacea septi*; but it was not possible to follow the branches for more than a few millimetres, as they appeared to break up and mingle with the cardiac muscle fibres. In

Figures V to X the large pale Purkinje cells, with their vacuoles, round nuclei and longitudinal striations, may be seen. They afford a striking contrast to the surrounding muscle fibres.

In the region of the *pars membranacea septi*, where the main bundle divides, there is normally a large amount of fibrous tissue, which makes it difficult to recognize a pathological fibrosis. Sections were taken from hearts with known branch bundle block; but it was impossible to conclude that in this small area the fibrous tissue was pathological. But in two cases of complete heart block we were able to demonstrate fibrosis and calcification of the main bundle of His.

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I should like to thank Professor Wingate Todd, of Cleveland, Ohio; Professor Keith Inglis, of the University of Sydney, and Mr. Murray, of the Kanematsu Institute, Sydney Hospital, for their help and cooperation.

DERMATITIS EUTORULOSA, WITH A DECRIPTION OF THE CAUSATIVE FUNGUS.

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DURING recent years a number of occasional papers on dermatitis due to yeast-like fungi have appeared in the journals. Though this is so, it is a peculiar fact that in none of these communications is a true yeast, a fungus of the family Saccharomycetaceæ, the subject of discussion; for the most part the communications deal with strains of Monilia, and such true yeasts as are mentioned are not pathogenic species.

The number of species of yeasts mentioned by Dodge[5] as having been regarded as causative of dermatitis is quite small and includes the following: *Debaryomyces fabrii* Ota and its variety *tremenoniensis* Ota, *Debaryomyces lundsgaardi* Ota, *Debaryomyces lædegaardi* Ota, *Debaryomyces gruetzii* Ota, and *Debaryomyces brunieri* Ota, *Saccharomyces sternoni* Sartory, and *Saccharomyces labialis* Ribeyro.

Of the imperfect yeasts the following have been regarded as causing dermatitis: *Cryptococcus psicrofiliacus* Nino, *Torulopsis mitis* Mazza, *Torulopsis mucilaginosa* Jörgensen variety *plicata* Lodder, *Eutorula furca* Fontoynont and Boucher[9] and *Eutorula bernasconi* Fontoynont and Boucher.

The pathogenicity of few of these species has been definitely established. In most instances they have been convicted on the slender evidence of their presence on the skin as shown by cultures from skin shreds or from the discharge. Both *Debaryomyces* and *Cryptococcus* contain definitely pathogenic species, for they have been conclusively proven to be responsible for affections clinically recognized as torulosis and blastomycosis.

In the case of *Debaryomyces gruetzii* Ota, the organism was isolated from the nail of a child affected with mycosis. The fungus caused death in twenty-six days, when injected into a mouse, and was recovered from the lesions found in the mouse. *Saccharomyces sternoni* Sartory was isolated from an interdigital mycosis and was said to produce similar lesions on the skin of a guinea-pig. *Saccharomyces labialis* Ribeyro was recovered from lesions on the lower lip of a young woman and was stated to cause abscesses in a guinea-pig. The pathogenicity of *Cryptococcus psicrofiliacus* Nino, *Torulopsis mitis* Mazza and *Eutorula bernasconi* Fontoynont and Boucher, was also fairly well demonstrated; but in no case was the organism subjected to the rigid test of Koch's laws.

It would appear then that at present it may be stated that six yeasts have been shown to be pathogenic and that they probably cause dermatitis, whilst another eight species have been accused of doing so.[1]

The present communication presents, it is believed, the first account of a yeast which is definitely capable of producing dermatitis; for the organism has been tested and found to fulfil Koch's laws. But before proceeding to the actual work in hand, I may perhaps be permitted to digress briefly in order to enter an emphatic protest against the looseness of clinical nomenclature of the fungi. Such is this looseness that much of the work done and its published accounts are more or less completely deprived of usefulness. This sterilization of the work results from the absence of definite citation of the authority for specific names and the absence or paucity of description of the morphological characters of the fungi dealt with.

Doubtless Castellani introduced untold confusion into medical mycology with his disregard of botanical standards of taxonomy and nomenclature. Unfortunately few clinicians have had any training in taxonomy at all, so that in following Castellani they were scarcely blameworthy. Even so it is difficult to understand how men like J. H. M. Macleod,[2] Stoval and Buloz,[3] G. B. Dowling,[6] and Stone[7] and Garrod,[1] working in well-known institutional laboratories, should have failed to give definite indication of the specific identity of the fungi with which they were dealing. More recently than these, Benham and Hopkins[4] contributed an exceedingly useful article on "Yeast-Like Fungi", giving adequate descriptions of the morphology and biochemical properties of certain of their species, which enable one to recognize with confidence those thus adequately described. I ask, nevertheless, why not have included the authorities for their

[1] Dodge does not include *Saccharomyces cerevisiæ* Meyen amongst the pathogenic species; but under particularly favourable conditions it can cause trouble. A young man received a burn, about one inch by two inches in area, on the front of the left forearm. This blistered and the blister burst. To relieve the pain the raw surface was wrapped in a cloth saturated with ale. Three days later the surface presented the appearance of a highly vascular weeping granuloma. Microscopic examination revealed yeast cells, and these on culture were identified as *Saccharomyces cerevisiæ*. A light curretting of the surface, followed by application of *tinctura iodi mitis*, cleared up the trouble rapidly.

specific names? The names *Monilia albicans*, *Monilia candida*, *Monilia parapsilosis* and *Monilia krusei* have all been applied to different species by different workers. Since Benham and Hopkins were in a position to compare their strains with those of the American national collection of standard cultures, it would have been of value to us to know that these species, so fully described by them, were identical with the species classified by Dodge[8] as *Syringospora albicans* Robin, *Monilia bonordeni* Vuillemin, *Mycocandida parapsilosis* Ashford and *Candida krusei* Castellani. In all probability this is the correct interpretation of their nomenclature; but we cannot be certain; the citation of the four specific authorities would have left no doubt. At long last an experienced taxonomic botanist, and one whose dicta must be treated with respect, has come to the assistance of the clinician. In his "Medical Mycology" Dodge has presented us with a complete classification of the fungi which are, and which have been stated to be, pathogenic. Clinical writers should remember that there is one way, and only one correct way, in which any specific fungus may be definitely and certainly cited, and that is by the quotation of both the generic and specific names of the fungus, and also, and just as essentially, the name of the author of the specific name. Once a species has been so indicated it may be referred to in subsequent discussion by a shorter title.

Prior to the appearance of Dodge's work, confusion was such that specific nomination and accurate citation were difficult, in many cases impossible to any but the experienced taxonomist. Since Dodge's book makes it relatively easy in most cases, and possible in every case, there is no longer any excuse for loose reference.

The names "moniliasis" for infection by various of the *Eremascaceæ imperfectæ*, "torulosis" for infections by various fungi not *Torula*, and "blastomycosis" for infections by fungi not *Blastomyces* are so deeply entrenched by common usage that, although botanically quite misleading, it were probably foolish to suggest that they should be replaced by more fitting names.

Eutorula Dermatitis.

Eutorula dermatitis is a localized papulovesicular and/or excoriated and weeping inflammatory condition of the skin, due to invasion of the skin by *Eutorula excorians* Kesteven, and causing more or less intense itching.

The condition was produced by the simple method of "patch testing" upon the flexor surface of my own arm, using portion of a pure culture obtained from Case X described below. From this experiment it is possible to describe the early course of the disease; the more advanced conditions, seen clinically, are detailed in the brief case descriptions below.

Twenty-four hours after the organism was rubbed on the arm the area treated was found to be definitely hyperæmic and felt warm. The following day a crop of fifteen to twenty papules had developed upon a hyperæmic area, and that area was now very irritable. The least rubbing caused it to itch intensely for about half an hour. Throughout the third day the area itched continuously and it was observed that most of the papules had become converted into vesicles. On the fourth day it was continuously itching and one of the vesicles apparently contained pus. This was opened with a sterile bistoury and the droplet of pus was spread upon a slope of Sabouraud's glucose agar. The tops of several of the vesicles were removed and placed on an agar plate, and the area on the arm was then treated with a 1% solution of dihydroxyanthranol in benzol. The following day the irritation was less and from then on the trouble cleared up rapidly under continued treatment. The photograph (Figure I) was taken on the fourth day.

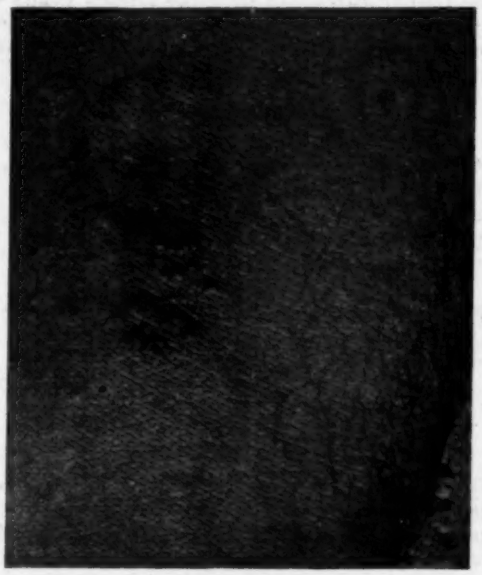

FIGURE I.
Appearances of experimentally produced eutorula dermatitis on the fourth day of infection.

Reports of Cases.

CASE I.—W.N., an adult male, complained of no symptoms at all; but it was found that he had sodden white skin between the little toe and fourth toe of each foot. Microscopic examination of the skin scales revealed no fungi; but the fungus was cultured from them, in association with *Staphylococcus albus* and two species of *Penicillium*.

CASE II.—N.B., an adult male, complained of "surfer's toe", which itched badly, especially at night. Between the little toe and fourth toe of each foot, and extending into the furrow beneath both toes, the skin was white and sodden. In places the horny layer had been shed, exposing the *rete Malpighii*.

CASE III.—G.L., an adult male, complained of "surfer's foot", with slight irritation, especially at night. The condition was similar to that in Case II.

CASE IV.—C.A.W., an adult male, complained of itching blisters on the side of his foot. Below and in front of the external malleolus of the left ankle there were three flat blisters, each about 1·25 centimetres (half an inch) in diameter, and nearly circular. Two of these had already broken, and the mucous layer of the skin was covered by a loose scale of the horny layer. The third was found to contain a watery white clear fluid. As in the previous cases, microscopic examination of the skin scales revealed no fungi; but the organism was found in smears from the fluid and also, as in the other cases, was cultured from the scales.

CASE V.—Mrs. G. complained of scalding on micturition and itching between the thighs. The whole of the con-

tiguous surfaces of both legs and the *labia majora* presented the appearances of an acute intertrigo, deeply injected and more or less completely denuded of the horny layer. The affected area was not sharply delimited, but presented a narrow transition band in which the hyperæmia faded away. The whole of the area was bathed in a white discharge, which in places had accumulated to resemble a streak of cream. No scales were obtained for examination; but the fungus grew in pure culture, without any contaminants, from the smears made from the discharge.

CASE VI.—J.S.H., an adult male, complained of scalding on micturition and rawness and itching of the skin on the scrotum and the thighs on either side of it. Examination revealed a patchy excoriated and extremely hyperæmic intertrigo extending to the sides and back of the scrotum and backwards up the intergluteal fold to above the anus. Quite extensive patches were completely denuded of the horny layer, exposing the weeping mucosa. The discharge was clear and slightly glairy. The trouble was sharply limited by a narrow fringe of white, sodden, desquamating *stratum corneum*. The fungus was found in the scales from the margin and was cultured in association with *Staphylococcus aureus* and *Staphylococcus albus* as well as a a species of *Penicillium* and a species of *Alternaria*.

CASE VII.—R.H., an adult male, was not seen until well on the way to recovery. Apparently the condition had resembled somewhat that described in Case VI. When seen the scrotum was found to be injected and to present a fine scaly desquamation. There was no denudation, and the slightly greasy nature of the surface was due in the main to a zinc cream which had been applied. The fungus was grown on Macleod's medium[6] in association with *Staphylococcus albus* and an *Aspergillus* species. This case resembled a "pityrosporic" intertrigo, hence the use of Macleod's medium.

CASE VIII.—J.C., an adult male, complained of an itching rash on his limbs. The condition was a chronic papulo-vesicular eruption on both thighs and legs, the upper surfaces of both feet, and to a lesser extent upon the flexor surfaces of both forearms. On the legs there were patches of fissuring and desquamation of the cuticle, with the formation of scabs. The fungus was not found in the scales, but was cultured from them in association with *Staphylococcus albus*, an *Aspergillus*, a *Penicillium* and a *Gliocladium* species.

CASE IX.—J.L., an adult male, complained of very irritating, itching and smarting sores on the hand. Both hands presented excoriated weeping sores of varying size. The trouble was most extensive on the palms, but extended to the interdigital spaces, the sides of the fingers and thumbs, the thenar and hypothenar eminences and the flexor surfaces of the wrists. The excoriated areas were surrounded by fringes of sodden desquamating cuticle or by groups of pinhead-sized vesicles. These slightly raised vesicles were found to contain a clear glairy fluid; a few apparently contained pus. The fungus was found in the caps of the vesicles and was grown in association with *Staphylococcus albus* and a species of *Penicillium*.

CASE X.—J.B., an adult male, was in a condition similar in all essential features to that of the last patient. The fungus was found in the caps of the vesicles and was grown in association with *Staphylococcus albus* and a species of *Alternaria*.

CASE XI.—C., an adult female, complained of a chronic sore around the nail of the middle finger of the right hand. The appearance was that of a chronic staphylococcal paronychia except that there was not the crusting usual in that condition, and the discharge was watery. The skin around the root of the nail was swollen as in a monilial paronychia. The fungus was cultured from the scales, in association with *Staphylococcus aureus*, *Staphylococcus albus*, two species of *Penicillium* and a species of *Cephalosporium*.

CASE XII.—J.C., an adult male, was in a condition essentially similar to that in Case VI. The fungus was cultured from the scales, in association with a species of *Aspergillus*.

CASE XIII.—V.C.R., an adult male, complained of "surfer's foot". This case was of interest because, though it was quite a mild condition of "surfer's foot", the fungus was found in association with *Epidermophyton niveum* McCarthy, as well as a species of *Aspergillus* and one of the *Eremascaceæ imperfectæ*.

Diagnosis.

It is doubtful whether the condition can be diagnosed clinically. The intertrigo in Case VI presented no feature wherein it differed from a typical staphylococcal intertrigo. On the other hand, the vesicular form of the malady resembles closely vesicular epidermophytids, more especially on the palmar surface of the hands, where the erythema is but slight. Whilst it cannot be asserted that they are pathognomonic, it may at least be said that the association of vesicles and/or a sodden cuticular fringe with excoriations should cause one to suspect the presence of this fungus. In the case of trouble between the toes, however, only an appeal to the agar plate will decide the question; for infection here by any one of the Trichophytonaceae and by apparently several members of the *Eremascaceæ imperfectæ* will give rise to precisely similar appearances.

Treatment.

Fortunately the condition yields readily to treatment. In my own experience the most effective application is 1% dihydroxy-anthranol in solution in benzol. As is well known, this substance is apt to produce untoward results on some skins, and must be used with caution. Eusol applied continuously for forty-eight hours is commonly effective, especially if the vesicles are carefully opened. Para-dinitrophenol may also be used in 1% aqueous solution; it is not so rapidly effective as either of the other two drugs, but is safe in all cases, and, as it needs painting on only twice a day, is more convenient than the eusol. Tincture of iodine is as effective as the dihydroxy-anthranol, but commonly causes an almost intolerable itching for several hours. It is not effective unless the vesicles are opened.

Before I proceed to a description of the fungus, a definite statement of the proof of its pathogenicity is desirable.

Proof of Pathogenicity.

The hairless area of the flexor surface of my own left forearm was firmly scrubbed with rectified spirit till a mild erythema was produced. The area was then further cleaned with ether. Two platinum loops full of the fungus from a pure colony obtained from Case X were then firmly, but not roughly, rubbed into part of the prepared area with a sterile gloved finger tip. The area was now covered with a piece of sterile "Cellophane", and that kept in place with a broad band of "Elastoplast" bandage. The resulting papulo-vesicular eruption has already been described. Subsequently the fungus was found growing, without contaminants, from the smear of the droplet of pus and from three of the caps from

vesicles. Throughout the whole of the work, identity of morphology has not been accepted as final proof of actual identity of the fungus; but every time the organism has been grown from scales or smears the resulting colony has been tested as to its cultural appearance on the various media, and as to its ability to liquefy gelatin and ferment the selected range of sugars. This procedure was adopted with the colony grown from the pus smear from my own arm, and the fungus proved in all respects identical with that grown from the twelve cases described.

There can, therefore, be no doubt that the fungus here described is capable of producing a papulo-vesicular dermatitis, and there is no reason to doubt that if not treated early such a condition, so caused, will progress to the excoriated condition seen in so many of the cases cited.

Eutorula Excorians, Species Novis.

Microscopic Appearances.

The cells of the many yeasts are so alike that there is little that can be said in a diagnostic vein of any of them. In this instance the predominating size is between 8·0μ and 10·0μ, and the variation observed is from the tiny newly-budded cells of 2·0μ up to large hyaline or diffusely stained, apparently dead and dying cells of 12·0μ. The cell of the predominant size has an eccentric nucleus, which may or may not be slightly lobed, and which takes a deep, even colour from the blue of diluted Giemsa stain. The cytoplasm is clear and may contain fine basophile granules. The cell wall is thin but is quite clearly definable. No acidophile substance is present in any of the cells; for no trace of the eosin is to be seen. As the cells enlarge the appearance is as though the nuclear substance were diffused throughout the cytoplasm. The nucleus appears to enlarge and stain less and less deeply. One finds a small minority of the cells around 10·0μ in diameter, no nucleus being definable, but the whole cell being filled with basophile granules. In still larger cells the stain becomes lighter, till there is a diffuse light blue through the whole cell. Finally, many of the largest cells are quite clear and transparent (Figure II).

The most careful search through many fields, both of living cells and preparations fixed with glacial acetic acid and stained with dilute Giemsa stain, has failed to reveal anything which might be interpreted as an ascocarp or ascospores. Cultures have been allowed to dry out slowly on agar on plaster to test for spore formation.

Simple binary fission in all its stages may be commonly seen in most microscopic fields. The commonest type is by the budding off of a small cell, 2·0μ in diameter: but equatorial and equal division also is not uncommon. The intermediate stages of subequal fission are also to be found. In all instances it has been the cell of the predominant size which has been observed dividing.

Macroscopic Features of the Colonies.

The organism grows freely on Sabouraud's glucose and maltose peptone agars, on Macleod's medium and on glucose, and maltose-peptone-gelatin media. In every case the appearance of the colony is the same. From a single point implantation the colony is nearly circular, smooth and shining, pure white in colour, recalling the polished surface of a piece of white ivory. There may, however, be faint radial and concentric striations. The centre of the colony is very slightly higher, deeper or thicker, than the margin. The colony does not grow continuously, but ceases to increase in diameter after the first four or five weeks (Figure III).

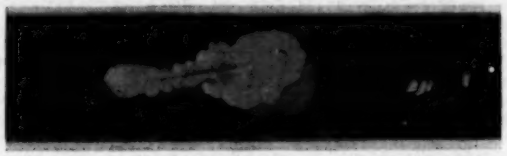

FIGURE III.
Showing the appearances of the growth of *Eutorula excorians* Kesteven on Macleod's medium.

When tested by lifting of a loopful, it is found that the growth is neither adherent nor coherent; if the loopful is put into water the cells may be very readily distributed evenly through it. The whole of the growth is upon the surface of the medium.

In all these colonies, grown on whatever medium, there is always a faint but quite distinct odour, recalling that which pervades a brewery, not exactly that of ale, but rather of fermenting hops.

The fungus was grown in fluid media, with a variety of sugars to test its powers of fermentation. In each instance the sugar was added to make a 4% solution in water, to which had been previously added 1% of Witte's peptone, 0·3% of sodium chloride and 0·5% of sodium hypophosphite. These tests enabled me also to observe its growth in fluid media. There was no development of a definite surface layer, and there was very little sedimentation at the end of two weeks. The following sugars were used in the test: arabinose, dextrin, glucose, inulin, lactose, mannite, raffinose, soluble starch and sucrose. Fermentation of the dextrin was the most abundant and rapid; that of glucose was second; mannite was third, sucrose fourth.

FIGURE IV.
Showing the appearances of a gelatin stab culture of *Eutorula excorians* Kesteven.

In gelatin stabs the fungus shows only faint streaks along the lines of stab and slowly liquefies the gelatin from the surface downwards (Figure IV).

Classification.

The complete absence of mycelium at once places this fungus as one of the Saccharomycetaceæ; the

absence of endospores excludes it from the amycelial group of the Coccidioideaceæ, whilst the absence of ascospore formation marks it as being one of the imperfect saccharomycetes.

Of the eight genera assigned to this group by Dodge, the new species conforms more nearly to the characters of *Eutorula* than any of the others, and it has accordingly been assigned to that genus. Of the three species of *Eutorula*, the present most nearly resembles *Eutorula bernasconi* Fontoynont and Boucher,[9] differing from it in that very few of the cells are pyriform, no short filaments whatever have been seen, and the cell membrane is delicate, not giving a double contour to the cells, as described by Fontoynont and Boucher.

Acknowledgements.

I have to thank Dr. F. C. Florance and Dr. C. F. Robinson, who have made most of the cases available to me and permitted me to report on them. I have also to acknowledge gratefully grants from the directors of the Goodyear Tyre and Rubber Company (Australia), Limited, with which my laboratory equipment and supplies were purchased.

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INFARCTION AND FIBROSIS OF THE HEART WALL.

By J. B. CLELAND, M.D.,

Marks Professor of Pathology, University of Adelaide; Honorary Pathologist, Adelaide Hospital.

IN 3,700 *post mortem* examinations performed at the Adelaide Hospital and the Mental Hospital, Parkside, nine examples of recent infarction of the heart wall, probably attributable in all these cases to atheroma, were found. In one case, that of a woman, aged thirty-eight years, the youngest subject, atheroma was noted in the coronary vessels, though it was not recorded in another woman, aged thirty-nine years. In addition, two examples were found of infective infarction in males, aged thirteen and seventy years, and one example was found in which infarction was thought to be due to an embolism in a woman aged fifty years. This gives a total of 42 cases, or a little more than 1%.

The distribution of the 39 examples attributed to thrombosis or atheroma was as follows. Twenty-one occurred in males, of whom one was aged forty-two years, five were fifty years and over, four were aged sixty years and over, seven were seventy years and over, three were aged eighty years and over, and in one case the age was not recorded. Eight examples occurred in women, of whom one was aged thirty-eight years, one thirty-nine years, one forty-eight years, two were fifty years or over, eight were sixty years or over, four were seventy years or over, and the age of one was not recorded. Of the total 36 cases, three subjects were aged under fifty years, seven were fifty years or over, ten were sixty or over, eleven were seventy or over, three were eighty or over, and the age of two subjects was not recorded.

There were six examples of rupture of the infarcted area with hæmopericardium. They occurred in males, aged sixty-two, seventy-seven and eighty-seven years, and in females, aged sixty-four, sixty-eight and sixty-nine years, and in one subject whose age was not recorded and in whom multiple ruptures were found. In addition, hæmorrhagic pericarditis was found in a man, aged eighty years; a woman aged sixty-six years had blood-stained fluid in the pericardium; and a woman, aged fifty-seven years, had ruptured a papillary muscle, part of which had been protruded through the mitral valve.

In the 3,700 *post mortem* examinations there were 26 examples of extensive fibrosis of the cardiac muscle attributable either to the gradual narrowing of the calibre of the vessels from atheroma or to actual thrombosis on an atheromatous patch. This gave rise to an infarcted area from which there had been a recovery, with conversion of the muscle into fibrous tissue. Two subjects were males and four were females. Of the 21 cases occurring in males, a man aged twenty-four years had Stokes-Adams syndrome as a result of the fibrosis, which was not associated with atheroma. A man of twenty-seven had cerebral thrombosis and thromboses in the descending aorta and carotid artery, and had presumably also had a thrombosis in a branch of the coronary artery causing the fibrosis. In males, aged thirty-six and forty-one years, coronary atheroma was not noted; but it was noted in a man of thirty-nine. A man, aged fifty-two, had heart block resulting from his fibrosis; but coronary atheroma was not noted. Coronary atheroma was noted in the two other men more than fifty years of age. In the remaining subjects, eight being over sixty, two being over seventy, three being over eighty, and the age of one not being noted, atheroma was recorded or was probably present. The four females were aged fifty-eight, sixty-three, seventy-six and seventy-seven years, and probably all had coronary atheroma.

Reports of Cases.

IN SEARCH OF A BROKEN PNEUMOTHORAX NEEDLE.

By M. P. SUSMAN, M.B., Ch.M., F.R.C.S. (England),
Honorary Assistant Surgeon, Sydney Hospital;
Honorary Thoracic Surgeon, Mater Misericordiæ Hospital, Sydney.

A HOUSE surgeon recently had a misadventure that has happened to the most experienced of "phthisiologists" (what a word!): while he was giving a refill of air to a patient with an artificial pneumothorax the needle broke in the depths of the puncture.

I made a small incision over the site of the puncture, but could neither see nor feel the remnant of needle. Then I inserted a trocar and cannula between the ribs and passed a thoracoscope into the pleural cavity through the cannula. The piece of needle projecting into the chest immediately came into the field of vision close to the cannula; I then inserted another cannula about 5·0 centimetres (two inches) in front of the first one, and through it I was able to touch the needle with forceps while looking through the thoracoscope. But before I could grip the needle securely I succeeded only in displacing it, and it dropped into the pleural cavity out of sight; I failed to find it with the thoracoscope and humbly closed the small punctured wounds (made for the cannulas) with horsehair stitches.

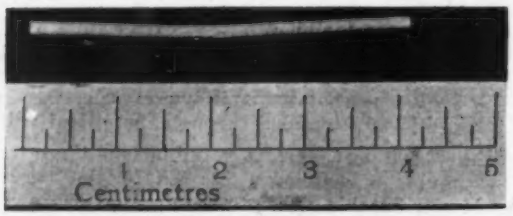

FIGURE I.
Showing the comparative size of the needle.

Skiagrams taken with the patient in different positions confirmed the fact that the needle was lying free at the bottom of the pleural cavity. With the patient on the sound side and inclined slightly backwards, I resected (under local anæsthesia) a segment of the tenth rib near the angle, and opened the chest through an incision 2·5 centimetres (one inch) long in the rib bed. After sucking out the small amount of fluid present (presumably due to the previous interference), I inserted a nine-millimetre bronchoscope and systematically examined the pleural cavity. The finding of the needle proved to be more difficult than I had expected, and it was not until I had raised the posterior edge of the collapsed lung with the beak of the bronchoscope that I saw it lying close to the costophrenic angle; it was now an easy matter to seize it with long biopsy forceps and remove it. I closed the wound with three layers of catgut and several horsehair stitches. The patient promptly recovered, and she was discharged from hospital four days later.

C. H. Andrews had a similar experience which he described in *The Journal of Thoracic Surgery*, Volume VI, Number 4, April, 1937. He was able to seize the needle through one cannula while looking through a thoracoscope inserted through a second cannula; then he lost his hold of it as he was trying to withdraw it from the chest. Fortunately, the needle was so situated that it was quickly found, grasped once again, and removed without more ado.

What is the best procedure for the removal of a broken needle from the chest wall or pleural cavity? Unless the needle end can be felt, screening and skiagraphy in several positions, or stereoscopic skiagraphy, are essential to decide whether it is in the chest wall or in the pleural cavity.

In the light of this, my own experience of the mishap, I offer the following suggestions:

1. If the needle is certainly free in the pleural cavity, its removal should be attempted with the help of a bronchoscope or thoracoscope, the site or sites of incision or puncture depending on the situation of the needle, as shown in skiagrams made with the patient lying on the sound side, that is, in the position which he will take on the operating table. The width of the intercostal spaces will decide whether rib resection is necessary.

2. If the needle is still apparently in the chest wall, I should make, over the site of puncture, a longer and deeper incision than I used here. If the needle is not thus found, I should deepen the incision still further, open the pleural cavity and insert a bronchoscope or thoracoscope; unless it has been dislodged, the needle may now be seen protruding into the pleural cavity and removed either through the original wound or through a fresh incision or puncture. If it is not to be seen thus projecting into the pleural cavity, we must presume that it has become dislodged, and is now right inside the chest. I should have new skiagrams taken and proceed as described in paragraph 1.

AN UNUSUAL CASE OF URINARY LITHIASIS.

By A. WALKER-SMITH, F.R.C.S. (Edinburgh),
Sydney.

THE patient, a man, aged fifty-three years, was referred to me on December 1, 1936.

History.

He complained of "passing blood in the water for three weeks". On investigation he was found to have a long history of urinary trouble, dating from a Neisserian infection contracted at the age of sixteen years. Two years after the initial urethritis, symptoms of stricture developed, and instruments were passed "a few times". The patient was quite definite that the urine had been cloudy at all times since his original trouble, and he stated that he had experienced occasional attacks of difficulty and increased frequency of micturition over a period of many years. An attack of hæmaturia occurred fourteen years before he consulted me, and since then he had passed blood in the urine on several occasions, culminating in the attack for which he sought treatment.

The symptoms present on the date of consultation were as follows: (i) hæmaturia, blood being intimately mixed with the urine; (ii) difficulty in micturition; (iii) frequency of micturition (he passed urine at half-hourly intervals during the day and had occasion to rise nine to ten times at night); (iv) pain at the end of micturition, referred to the perineum; (v) constant dull ache in the suprapubic region, worse on exertion. All these symptoms had been pronounced since the onset of hæmaturia three weeks before. The appetite was moderately good, thirst was not excessive, and there had been no loss of weight. The bowels were constipated.

Investigation.

The patient appeared to be a well-nourished man, who looked his age. He was rather pale and wore a worried look. The tongue was moist, but moderately furred. General examination elicited no abnormality apart from the genito-urinary system. He passed a very thin dribbling stream of urine, with great difficulty. The urine was

deep red in colour, and on microscopic examination many red blood cells and pus cells were evident, together with numerous motile bacilli and phosphates.

Great tenderness was found on palpation of the suprapubic area; but there was no apparent distention of the bladder. There was no tenderness in the loins and the kidneys were not palpable. A hard tender mass the size of a Brazil nut was palpable in the perineum in the vicinity of the bulb. On rectal examination a large, extremely hard mass was palpable in the region of the prostate. It felt round and regular and was considerably larger than a golf ball; it was not tender. It was felt to be immobile on bimanual palpation. A urethral bougie would not pass beyond twelve centimetres.

The patient was admitted to hospital on December 3, 1936. The blood urea content was found to be within normal limits, namely, 36 milligrammes per 100 cubic centimetres of blood. X ray examination of the urinary tract showed no abnormality in the renal areas; but in the film of the lower part of the tract the condition seen in Figure I was presented. The large pear-shaped shadow was thought to be a calculus in the prostatic urethra, while the distal opacity obviously was a stone in the bulbous urethra. The shadow superimposed on the large opacity was considered to be a vesical calculus; but whether it was connected to the large calculus, forming a vesico-prostatic stone, could not be foretold.

Operation.

Operation was performed on December 7, 1936, under ether anæsthesia. The bladder was opened and found to be greatly hypertrophied. A soft phosphatic calculus a little smaller than a golf ball was removed, together with much phosphatic débris. The internal sphincter was inspected and appeared to be normal; it was manually dilated, and the large stone was palpated. A "V" was then cut from the posterior lip of the internal meatus, and it was found that three faceted stones were present instead of a single large calculus, as I had imagined. This fact facilitated the removal of the offending mass, as, one stone having been extracted after some manipulation, there was little difficulty in removing the remainder.

The prostate was thinned out to a mere shell, so the cavity was curetted and the bladder was closed round a large suprapubic tube. The patient was now placed in the lithotomy position and the urethral calculus was removed by perineal section. This stone also was found to be composed of four beautifully faceted pieces. A stricture just distal to the site of the calculus was dealt with, and three interrupted sutures of fine catgut were inserted in the urethral incision. The skin wound was not sutured.

After-Treatment.

The suprapubic tube was left *in situ* for three weeks in order to allow the urethra to heal soundly, and before its removal a metal bougie (size $^{12}/_{10}$, English) was passed *per urethram* without difficulty. The patient was discharged from hospital on January 16, 1937, when the wounds were soundly healed and he was passing urine freely.

Results.

The patient was last seen on September 23, 1937, when he passed urine with a normal stream. A catheter entered the bladder easily and there was no residual urine. The urine contained a few pus cells only.

The patient stated that there was some difficulty in holding the urine if the bladder became over-distended; but he was able to sleep through the night without disturbance.

Size and Composition of the Calculi.

The stones removed from the prostatic urethra were assembled and glued together. The resultant calculus weighed 90 grammes (three ounces) and measured 5·3 centimetres (two and one-eighth inches) by 5·0 centimetres (two inches) by 3·8 centimetres (one and a half inches). It was composed of oxalates with a trace of phosphates.

The stones in the bulbous urethra together weighed 30 grammes (one ounce) and measured 5·0 centimetres (two inches) by 2·2 centimetres (seven-eighths of an inch) by 2·2 centimetres (seven-eighths of an inch). They also were composed of oxalates with a trace of phosphates. The vesical calculus could not be preserved, as it was very soft and was composed of phosphates.

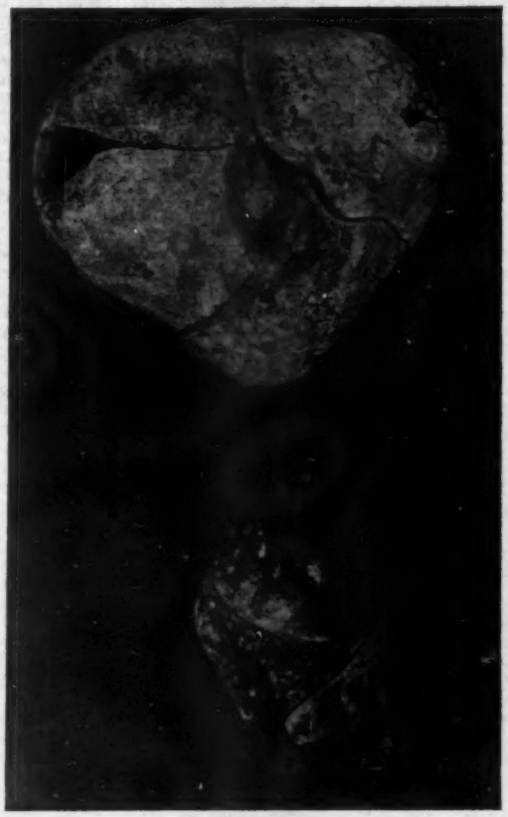

FIGURE II.

Reproduction of a photograph of the reconstructed calculi after removal.

Comment.

1. It is interesting to note that in spite of the extraordinary urethral obstruction the general health was reasonably good and the blood urea content was within normal limits.

2. The ætiology of the condition was doubtless a urethral stricture, following a Neisserian infection. A careful examination of the X ray film showed a definite nucleus at the distal end of the bulbar calculus, showing that the stone formation commenced at this point.

3. It is most unusual to find calculi in the bladder, prostatic urethra and bulbous urethra of the same individual at the same time, and it is rare to encounter a calculus, or rather calculi, of the dimensions described in the prostatic urethra.

4. Also I think it is rather surprising that a person should suffer distressing symptoms for such a prolonged period without seeking advice.

Reviews.

THE RADIOLOGY OF PULMONARY TUBERCULOSIS.

"THE RADIOLOGY OF PULMONARY TUBERCULOSIS",[1] by J. E. Bannen, is a handy and concise little book which deals in a clear manner with the modern radiological aspects of pulmonary tuberculosis and, in addition, contains an excellent chapter on the clinical aspects of the disease by Dr. Diamond, senior tuberculosis officer, Hull. The various chapters deal with the normal lung, technique of examination and the radiological appearances of childhood and adult tuberculosis; differential diagnosis, pneumonokoniosis and collapse therapy also receive consideration. The author stresses the necessity for good films, and recommends the postero-anterior film, taken at six feet distance, as being the most useful; oblique and lateral films are necessary only to clear up some obscure point. High milliampèrage (200) at 50 to 55 kilovolts for one-tenth of a second gives excellent pictures; higher kilovoltages give less contrast. Films are taken in deep inspiration. Tomography is of help in some cases after the particular lesion has been localized. The normal lung and the difficult hilar regions, with descriptions of the positions of the lobes, are dealt with in detail. The various inflammatory lesions are grouped under three headings: (*a*) exudative and proliferative, (*b*) fibrotic, (*c*) destructive; each gives a characteristic X ray appearance. The various skiagrams used to illustrate these conditions are very well reproduced, without any retouching. Early tuberculous lesions may be demonstrated long before there are any clinical manifestations, and when no tubercle bacilli are found, repeated X ray examinations will exclude the possibility of tuberculosis. Early foci may be missed and simple infective processes may cause confusion. The author sees no advantage in the use of the fluoroscopic screen. Primary infections (childhood type) are surrounded by exudative lesions, whereas secondary infections (adult type) are characterized by productive and fibrotic lesions, according to the state of allergy existing. Childhood infections are air borne and involve the alveoli, with resulting inflammatory reactions; a bronchopneumonic condition follows, with later fibrosis. The size of the lesion depends on the virulence of the infection. The tracheo-bronchial lymph glands are involved. Healing occurs, with calcification and encapsulation. Once calcification occurs any further infection must be of the adult type. Calcified areas may break down in later life and cause reinfection; in the adult type productive, exudative and fibrotic lesions are present. Adults show more pronounced constitutional signs than children. The author points out that in most cases a radiological diagnosis can be made, but that it is always better to combine radiological evidence with a thorough clinical examination. Tuberculous lesions have a tendency to calcify and show patchy distribution. The author states that no lesion should be considered as healed unless repeated X ray examinations over many months have been carried out; in fact, it is always better to refer to a lesion as being "arrested" rather than as being "healed". The degree of activity can be stated from the radiological examination alone.

The differential diagnosis is considered in Chapter VI, while Dr. Diamond's contribution on the clinical side should be read carefully by both radiologist and clinician. Dr. Diamond issues a warning against the giving of a positive pathological report of tubercle bacilli when only a few bacilli are noted; repeated examinations (at least three) should be made in all cases. A chapter on pneumonokoniosis is included, but contains no new matter; the author still leans to the possibility of Jones's sericite as being a cause of silicotic fibrosis. Collapse therapy under radiological control is of great value in selected cases.

This small book is the best on the subject that has been published for some time, and should be in the hands of every radiologist and every physician who deals with pulmonary tuberculosis.

[1] "The Radiology of Pulmonary Tuberculosis", by J. E. Bannen, M.B., Ch.B., D.M.R.E.; 1937. London: Baillière, Tindall and Cox. Demy 8vo, pp. 163, with illustrations. Price: 12s. 6d. net.

BIOCHEMICAL METHODS.

IF the number of text-books which are published from year to year is any indication, continually increasing importance is being attached to the instruction of medical students in biochemistry. Professor F. C. Koch's manual on practical methods[1] was originally prepared for use concurrently with Mathews's "Textbook" and his later "Principles of Biochemistry".

The manual is divided into three main parts: the chemistry of the cell constituents, the chemistry of digestion, and the chemistry of the blood and urine. The greater bulk of the book is devoted to the last part, which is mainly concerned with quantitative methods.

The first section consists of qualitative experiments on the carbohydrates, lipides and proteins. Rather a wider range of tests than usual is described. The instructions are distinguished by precision of detail. It is surprising to find no mention of the inorganic constituents of the cell in this section.

The section on digestion deals very fully with methods for the demonstration of the activity of the digestive enzymes. The conventional methods for the analysis of the gastric contents are described, but the fractional test meal is not mentioned.

The qualitative part of the section on blood and urine is disappointing. Among the blood pigments, for example, sulphæmoglobin is not mentioned, although its detection is now of some importance. The scheme adopted to show the relation between the blood pigments is an obsolete one. Among the tests for the detection of blood the now widely used Takayama reaction is not even mentioned. The qualitative section on urine is also disappointing. No methods of examination of urinary deposits and calculi are described.

The quantitative portion of the section dealing with the blood and urine is more comprehensive. In fact the amount of work in this part is far beyond the scope of a student taking the ordinary medical course. Many of the methods described require a greater experience of biochemical technique than can be expected of the third-year medical student. This portion of the work is much more advanced than the rest. The descriptions of the methods are very clearly given. A number of these methods, it should be noted, have been developed in the author's laboratory in Chicago. He states in his preface that in this, the second, edition a number of duplications of methods have been eliminated. Further improvement in this direction is desirable. For example, a description of Van Slyke's volumetric methods of gas analysis seems unnecessary when his more recent manometric methods are given in detail.

A full appendix supplies all the necessary data for the preparation of the reagents used in the various experiments. The manual should prove useful to students taking a course of biochemistry more advanced than is possible in the present medical curriculum.

[1] "Practical Methods in Biochemistry", by F. C. Koch; Second Edition; 1937. London: Baillière, Tindall and Cox. Super royal 8vo, pp. 311. Price: 10s. net.

The Medical Journal of Australia

SATURDAY, JUNE 4, 1938.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

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THE EMPIRE RHEUMATISM COUNCIL.

REFERENCE to the increasing realization by the medical profession and by leading industrialists of Great Britain of the ravages upon the health and working capacity of employees made by chronic rheumatism has appeared in these columns upon several occasions. The International League against Rheumatism can claim much credit for this change, while medical statisticians and insurance bodies in all countries have produced startling figures of the vast economic loss for which cardiac and skeletal rheumatism is responsible. At the instigation and with the continued encouragement of the Royal College of Physicians of London, a specialized and particularly well-informed medical committee publishes regular and valuable reports upon clinical aspects of the disease. The British Red Cross Society, anxious to provide practical benefits for the sufferers in both peace and war, has achieved remarkable success with its model clinic at Peto Place, London. A little more than a year ago the Empire Rheumatism Council took shape, and it has recently published its first annual report. This body, by virtue of its personal and financial backing, outshines all other British organizations which attempt to tackle the problem of rheumatism. Under the presidency of the Duke of Gloucester and the chairmanship of Lord Horder, most of the best-known physicians and specialists in the treatment of rheumatic disease have linked forces with the wealthy employers of London. A large sum of money has already been collected. The activities of the past year have been chiefly devoted to organization. Research fellowships are to be made available at Peto Place and in the large metropolitan hospitals of London. At provincial spas, where already so much good research is in progress and clinical benefit obtained, the influence and aid of the Council will become apparent. It is proposed that special rheumatic wards in the larger hospitals, rather than separate rheumatic hospitals or clinics, should be established. It is evident that the governing authorities of the larger hospitals are prepared to help to the utmost.

A scheme of this magnitude is certain to relieve the sufferings of the rheumatic cripple. It is of the same order as the Empire Cancer Campaign, but with a more soluble problem to face. No doubt the question of prophylaxis in certain occupations and in predisposed or threatened persons will receive close attention. The rheumatic sufferer will no longer fall between the two stools of medical and orthopædic treatment. He will come under the care of a specialist who is himself an authority on all aspects of the rheumatic problem, medical, surgical and social. The facilities for treatment will include the whole resources of physiotherapy and hydrotherapy and all the opportunities for investigation provided by a large hospital.

It is hoped, and it is the written desire of the Empire Rheumatism Council, that an affiliated body will be formed in Australia to pursue here the war on rheumatism. The formation of such a body has already been advocated, and the National Health and Medical Research Council has stated its intention of immediately prosecuting research into rheumatic disease in this country. It is to be hoped that cardiac rheumatism will be included with joint and

fibrous tissue rheumatism in such an attack. Surely the insurance companies, which have already seen fit to appoint medical officers and to provide dressing stations for certain purposes, should combine for their own benefit, if for no other reason, with medical and other bodies which are attempting to assuage the rheumatic curse. The problem is far-reaching and requires considerable funds and authority for its pursuit. The Australasian Medical Congress to be held in Perth in 1940 may hold a plenary session on rheumatism. But this event is too far ahead. Preliminary ground work for a concerted effort which will have the backing of the Empire Rheumatism Council is required immediately. A great opportunity for service and prestige awaits the metropolitan hospitals in Australia whose governors first have the foresight and sympathy to establish a special ward for the investigation and treatment of rheumatism.

Current Comment.

THE PHARMACOLOGY OF PHENOLPHTHALEIN.

IN view of the immense consumption of phenolphthalein as a purgative, it is fitting that its pharmacology should be accurately elucidated. The purgative action of this drug was discovered by Z. von Vamossy, who discovered in 1902 that over 85% of the quantity ingested was excreted in the fæces of dogs, and that only a very small amount of free phenolphthalein could occasionally be found, on the addition of an alkali, in the urine. This investigator also demonstrated in experiments on dogs that there was an increase in the conjugated sulphates in the urine after administration of very large doses. Bernard Fantus and J. M. Dyniewicz have conducted an extensive study of the elimination of this drug from the body.[1] They state that Kastle was probably the first to discover the presence of conjugated phenolphthalein in the urine. After administering 0·5 gramme of phenolphthalein intraperitoneally to a guinea-pig, he noted the excretion of conjugated phenolphthalein in the urine for twenty days. M. C. Fleig concluded that phenolphthalein was so stable that it was not broken up into phenol and phthalic acid in the system. After large doses are given orally it passes in the free state into the urine. It also causes an increase of the conjugated sulphates in the urine. This increase is particularly marked when injections of the drug are administered beneath the skin or into the blood. A small quantity of conjugated glycuronate was also found in the urine of dogs after ingestion of large amounts of phenolphthalein.

Fantus and Dyniewicz had ascertained, in a previous study of urinary elimination after one thousand medicinal doses of phenolphthalein, that free phenolphthalein was generally absent from the urine when small medicinal doses had been given, and that the larger the dose, the more often did free phenolphthalein appear in the urine. Conjugated phenolphthalein was always present. Consequently a test of the urine for conjugated phenolphthalein would suffice to show when phenolphthalein had been administered. The estimation of the amount of free and of conjugated phenolphthalein in the urine may suffice to indicate the excretion of total phenolphthalein in the urine. Fantus and Dyniewicz now report the results of careful quantitative estimations of the total amount of phenolphthalein eliminated in the urine after a medicinal dose by continuing the determinations of the amount contained in successive twenty-four hour specimens until it was no longer demonstrable in the urine. It was considered that such a method might form a basis for the determination of abnormalities of elimination and might also provide an index of the value of methods of treatment after overdosage with phenolphthalein. Fantus and Dyniewicz describe their methods of determining conjugated and free phenolphthalein in the urine. They were able to report on the results of experiments to determine the total elimination of the drug after the taking of fifty-five doses by ten different volunteers who were thoroughly cooperative. Attention was concentrated on the influence of certain factors on the total quantity of phenolphthalein eliminated. These factors were cathartic action of the drug and size of the dose, the method of its administration and the degree of elimination on successive days after ingestion of the dose. When the investigators studied the relation of conjugated phenolphthalein elimination to cathartic action, the interesting fact emerged that the presence or absence of cathartic action had a definite determining influence on the urinary elimination of phenolphthalein. They state that there is an inverse relationship between the two. Hence the greater the cathartic action, the less is the urinary excretion of phenolphthalein, and *vice versa*. Further, the results show that the quantity excreted bears no proportion to the amount administered. In general, the greater the dose, the smaller the percentage eliminated. The apparent paradox may be explained when we consider that the greater the dose, the greater will be the cathartic effect. Accordingly, more will be lost in the fæces and less will remain for elimination in the urine.

A suggestion has been made that this purgative should be administered in "broken dosage" in the same way as calomel, in order that a more pronounced effect might be gained by better utilization of the solvent available. This expected result did

[1] *The Journal of the American Medical Association*, March 12, 1938.

not necessarily follow. It was found that in some cases "broken dosage" resulted in absence or diminution of cathartic effect, with a greater urinary excretion of the phenolphthalein. When the elimination of phenolphthalein in the urine over a period of twenty-four hours was plotted after the administration of several doses, it was found that, although the curves varied in detail, they were in agreement in one respect, namely, that elimination attained its peak in the second twenty-four hours, and in most cases terminated in the fourth twenty-four hours. It was found that this was true irrespective of dose and method of administration. In an occasional person elimination continued up till the sixth or even the eighth day. When this happened, no purgative effect had followed any of the doses.

As a result of their investigations Fantus and Dyniewicz conclude that free phenolphthalein is usually absent from the urine or only present in traces after the customary medicinal doses. On the other hand, it is constantly present after the exhibition of doses greater than 240 milligrammes. In such cases the total elimination of free phenolphthalein may reach from one to two milligrammes in twenty-four hours. Conjugated phenolphthalein is invariably present in the urine of persons who take phenolphthalein. The total amount of free and conjugated phenolphthalein excreted varies between 2·02 and 17·67 milligrammes when the dosage ranges from 60 to 480 milligrammes. The quantity of phenolphthalein eliminated varies between 1·41% and 19·7% of the amount consumed. The peak of elimination is attained on the first day in 44% of the cases and on the second day in 52%. In only 4% does the peak occur on the third day. It never occurs later than that time. The duration of elimination does not exceed six days as a rule, unless cathartic action does not occur. When catharsis is effected elimination usually terminates by the fourth day.

These studies form an interesting addition to our pharmacological knowledge. Other matters concerning phenolphthalein, however, still await elucidation. What is the exact explanation of the renal irritation which occasionally follows its administration? What explanation can be advanced for the very persistent cutaneous eruption, resembling *erythema multiforme,* which is sometimes noticed after its exhibition?

THE RELIEF OF PAIN IN DIABETES.

SEVERE neuritis of such a grade as to produce not only persistent pain, but also reflex and muscular disturbances, is not a very common complication of *diabetes mellitus.* The lesser grades of neuritic pain are by no means rare, and to these must be added the pains due to vascular ischæmia. The ætiology of diabetic neuritis has been a subject for argument for a long time. Some authorities have maintained that hyperglycæmia alone was enough to account for neuritis, though it must be admitted that there are very few cases of this complication in which vascular disease does not exist—a fact which makes it difficult to blame the high level of the blood sugar alone.

Sandstead and Beams have written recently on the use of sodium chloride for the relief of the neuritic pains of diabetes, and they describe thirteen well-studied cases in which this treatment was effective.[1] They assert that they were able to exclude hyperglycæmia *per se* and vitamin deficiency as causes of the neuritis, and, further, that the removal of septic foci, when such existed, caused no improvement. To what extent the neuritic disturbance was dependent upon vascular insufficiency Sandstead and Beams are unable to prove; but they believe that the results of treatment throw some light on the question. They also quote Woltman and Wilder, who, finding that neuritis commonly occurs in well-controlled diabetics, were able to demonstrate the existence of arteriosclerosis in the intraneural vessels at a number of autopsies. Dealing with the question of treatment, the authors recall that hypertonic saline solution has been recommended and used for some time past in vascular disease, for example, *thrombo-angiitis obliterans,* and even in coronary sclerosis. The oral administration of sodium chloride has also met with some success, and it was the object of Sandstead and Beams to present a series of cases of diabetics suffering from pain of vascular or neuritic origin in which satisfactory results had followed the use of this simple method. The details of treatment are free from complexity, for all that is necessary is to give to the patient a daily dose of 0·25 to 0·5 gramme of sodium chloride for each kilogram of body weight. If a solution of the salt is sipped slowly three or four times daily, it does not cause any gastric disturbance. It is given for a period of two to four weeks, followed by an intermission. In this way administration may be continued for a year if desired. No ill-effect has been observed, and complete or at least considerable relief from pain has been achieved.

The authors cannot explain the means whereby the salt produces the circulatory changes which apparently occur. It has been suggested that the alterations in diffusion and osmosis which take place, act as a sort of exercise in fluid interchange between vessels and cells; but they could demonstrate no variation in the electrolytes or other components of the blood after administration of the salt. Two patients showed a slight œdema of the feet; but no constant change in weight was observed. That a real improvement in circulation occurred was shown by the histamine test, and though no positive proof is presented, it seems certain that all such changes for the better hinge on the alterations in vascular conditions. The ease with which this treatment can be carried out and its apparent harmlessness commend it for trial.

[1] *Archives of Internal Medicine,* March, 1938.

Abstracts from Current Medical Literature.

BACTERIOLOGY AND IMMUNOLOGY.

Sulphanilamide and Antiserum in Meningococcus Infections in Mice.

THOMAS MCPHERSON BROWN (*Bulletin of the Johns Hopkins Hospital*, October, 1937) has studied the protective action of sulphanilamide and meningococcal antiserum on mice infected with meningococci. Two recently isolated strains of meningococcus were used and suspended in mucin. The minimum fatal dose was worked out to be 10^{-6} of the original broth culture. The mice were inoculated intraperitoneally with 0·5 cubic centimetre of the suspension, dilutions being made so that the doses received were from 10^{-7} to 10^{-10}, and the injections were given within half an hour of dilution in the mucin. Sulphanilamide solution of 0·8% strength was prepared according to the method of Perrin Long, and so diluted that the dose used was always in a volume of 1·0 cubic centimetre. This was injected subcutaneously immediately after the injection of the organisms. The antiserum used was a concentrated one, and dilutions were made so that the test dose was contained in 0·5 cubic centimetre. The first series of experiments showed that 8·0 milligrammes of sulphanilamide were effective in protecting mice from 100,000 minimum fatal doses of meningococci, 4·0 milligrammes protected against 10,000 minimum fatal doses, while 1·16 milligrammes were not effective. With larger inocula no protection was afforded. Blood cultures collected eight hours after inoculation were invariably "negative" in the mice which recovered. Experiments with serum alone showed that 0·1 cubic centimetre of serum protected 50% of animals against 100,000 minimum fatal doses, while 0·001 cubic centimetre was ineffective. A third series of experiments showed that the use of antiserum in conjunction with sulphanilamide afforded protection against inoculation with larger doses of organisms, and that these therapeutic agents were efficacious in smaller dosage when used in conjunction than when either was used alone.

Fibrinolytic Activity of Hæmolytic Streptococci.

ELIZABETH JOLLY, R. H. WEAVER AND M. SCHERAGO (*Journal of Infectious Diseases*, September-October, 1937) have studied the fibrinolytic activity of hæmolytic streptococci from normal and diseased throats. The normal group consisted of a series of repeated throat swabbings from a small number of medical students, and of single swabbings from a large number of outpatients. A high proportion of positive cultures was obtained. The method of plasma clot lysis by whole young cultures, elaborated by Tillett and Garner, was employed, and four strains included which were known to produce lysis within thirty minutes under the conditions of this method. The results showed that the strains isolated from the normal subjects were only partially and slowly fibrinolytic. Cultures were made from material from the throats of patients with low-grade infections of the upper respiratory tract. All these cultures displayed a much greater fibrinolytic activity, and in a shorter time, usually under twelve hours. Three strains were isolated from cases of scarlet fever, and these were the most active of all strains examined. Cultures from contacts of these cases who did not contract the disease showed weak and slow fibrinolysis, while the one contact who did contract the disease showed an active rapid test. Hæmolysin titrations were carried out on all strains, and there was no correlation between hæmolysis and fibrinolysis, nor was there correlation between the degree of hæmolysis and the severity of the condition from which the strain had been isolated. The authors suggest that the fibrinolysis test might be of value in the detection of "significant contacts" of cases of scarlet fever.

Sources of Error in Fermentation Tests.

N. E. GOLDSWORTHY, J. L. STILL AND J. A. DUMARESQ (*The Journal of Pathology and Bacteriology*, March, 1938) have studied the sources of error in the interpretation of fermentation reactions, with special reference to the effects of serum enzymes. The variable results obtained in the testing of the fermentation reactions of organisms requiring media enriched by the addition of horse serum, drew attention to the possibility that the irregular changes might be due to the presence of enzymes added to the media in the serum. Media sterilized by filtration gave many more positive tests than those sterilized by heat, and experiments were devised, samples of serum being used which had been heated at different temperatures for varying periods of time. It was found that the heating of serum for one hour at 65° C. before its addition to media was sufficient to destroy the serum enzymes, and fermentation reactions performed with such media gave constant results. The presence of other factors in serum which may interfere with the fermentation of carbohydrate added to the medium is also discussed.

Hæmolytic Streptococci in the Throats of Normal Adults.

ARTHUR W. FRISCH (*Journal of Infectious Diseases*, January-February, 1938) has studied the incidence of β-hæmolytic streptococci, their fibrinolytic activity and the occurrence of Group A strains in cultures taken from the throats of a large group of undergraduates, and has noted the presence or absence of tonsils. Cultures were made in beef infusion broth and sheep blood agar plates were used for isolating the hæmolytic streptococci. Agglutination tests were made with material supplied by Dr. Lancefield, and used according to her method, and fibrinolysin tests were made according to the technique of Tillett and Garner; controls with known strains were made throughout. The results showed that only 6·4% of 621 students carried Group A strains in their throats, although hæmolytic streptococci were present in 16·2%. Fibrinolytic strains numbered 10% of the total. Hæmolytic streptococci and also Group A strains were more frequently found in the throats of persons possessing intact tonsils, although this was a numerically smaller group. The number of fibrinolytic strains found was intermediate between the total number of strains of hæmolytic streptococci and the number of Group A strains. Repeated cultures were not made, so that the persistence of the carrier state was not investigated. The author comments on the findings of other workers who have shown that Group A strains are rarely present in the birth canal, and remarks that the presence of hæmolytic streptococci in the throats of carriers may well be the source of exogenous and autogenous genital tract infection.

Immunity to Tetanus.

P. A. T. SNEATH, E. G. KERSLAKE AND F. SCRUBY (*American Journal of Hygiene*, May, 1937) have tested the resistance of guinea-pigs treated by passive and active immunization to lethal spore doses of *Clostridium tetani*. A stock strain of *Clostridium tetani* was maintained in semi-solid agar culture and broth subcultures were made. These were heated in an Arnold sterilizer for thirty minutes, centrifuged, washed, and the sediment made up to a volume of 2·0 cubic centimetres with saline solution. These constituted spore emulsions from which serial dilutions were made and planted in tubes in volumes of 0·1 cubic centimetre; colony counts were made of the discrete growth obtained in the higher dilutions. These latter dilutions were stored and used for the experiments, although it was realized that there might have been some spore destruction. Spore tests were made by mixing equal volumes of the dilution chosen, of saline solution and of 50% calcium chloride solution, and injecting this preparation into guinea-pigs. In the first series of experiments the smallest amount of spore emulsion which would produce tetanus in the intact animal was determined, and colony counts showed that this lay in a dose of the serial dilution of 10^{-13} in an amount of 0·1 cubic centimetre, containing six to fifty spores. This was referred to in the subsequent work as the "lethal spore dose". A group of guinea-pigs were then subjected to passive immunization by a dose of 1,500 units of tetanus antitoxin given intraperitoneally directly after the "lethal spore dose". Of twenty pigs, five died of tetanus, five showed no

evidence of infection, and the remaining ten showed evidence of tetanus from which they recovered. With larger spore doses the amount of antitoxin given afforded no protection. Three groups of guinea-pigs were immunized by the injection of toxoid. The animals of the first group received three doses of toxoid at intervals of two weeks; those of the second group received one dose of 1·0 cubic centimetre of alum-precipitated toxoid, those in the third group received two doses of 0·5 cubic centimetre of alum-precipitated toxoid at intervals of four weeks. All three groups were then given the "lethal spore dose" previously determined. In the first group, five pigs died of tetanus, two recovered after showing symptoms, and fourteen evinced no sign of infection. Four animals of the second group recovered, and in the remaining eleven no evidence of infection was seen. In the third group no animal showed any signs of tetanus. The authors consider that these experiments show that active immunization by the administration of toxoid was more efficacious than the injection of single large doses of tetanus antitoxin.

HYGIENE.

Infection with Trichomonas in Heifers.

JUSTIN ANDREWS AND FRED W. MILLER (*American Journal of Hygiene*, March, 1938) have carried out an investigation of the presence of trichomonad infection in a properly supervised herd of forty heifers free from Bang's disease. Vaginal material was collected by aspiration, a blunt-ended piece of glass tubing being used, to which was attached a rubber bulb, the tube being inserted through a speculum after a preliminary scrubbing of the vulva. The bulls which were potent were invariably infected with trichomonads and infected the heifers. Ninety *per centum* of these calved normally after less than two services and with a delay of three weeks. These were all light infections. Of the heifers with heavy infections, only four calved normally, with a delay of a full period of gestation, and required ten services per animal. The remainder were infertile. The reproductive incapacity in heifers with heavy infections was striking. In these animals vaginal changes could be seen and uterine discharge was present. After the first calving the intensity of the infection and its results greatly diminish and possibly disappear.

Incidence and Distribution of Ascaris Lumbricoides in North Carolina.

A. E. KELLER, W. S. LEATHERS AND J. C. KNOX (*American Journal of Hygiene*, March, 1938) have observed the presence of *Ascaris lumbricoides* in 9·5% of 36,930 specimens of fæces. An infestation rate five times as great occurred in persons living in mountain areas and in swampy coastal areas. This is the reverse of the hookworm incidence. The incidence is greater in persons under fourteen years of age, and nearly twice as heavy in females. In coastal areas there was a heavy incidence among negroes (three times as great) and a heavier intensity of infestation as contrasted with the lower incidence in whites (7%) in these areas. In both sexes the peak of infestation occurs in persons between the ages of five and nineteen years. *Trichuris trichiura* occurred in fewer persons, but its district incidence and the frequency of its occurrence in negroes correspond to those of *Ascaris*. Little difference in intensity of infection was noted. *Hymenolepis nana* was far less common in all persons and was much less common in negroes. *Hymenolepis diminuta* was rare in both races.

Blood Platelets in Hookworm Anæmia.

J. W. LANDSBERG (*American Journal of Hygiene*, March, 1938) has made a series of observations on the thrombocytes in young dogs given fatal and non-fatal infections of the dog hookworm, *Ankylostoma caninum*. The platelets were enumerated by the direct method of Rees and Ecker, being stained with brilliant cresyl blue and counted directly with the red cells. The normal platelet figure for this group of young dogs was established as 154,879, with a standard deviation of 3,300. This figure is lower than those published by other authors working with dog hookworm. In the group which received fatal infections, there was a rapid lowering of the hæmoglobin value to one to four grammes per hundred cubic centimetres, while the platelets remained at or about the normal mean established on the uninfected animals. In the animals given non-fatal infections the hæmoglobin fell, and rose to normal with recovery, but there was no corresponding variation of the thrombocytes. The presence of an anticoagulant in the anterior part of the worm itself, the observation of rapid clotting at the puncture in the marginal ear vein, and the absence of marked change in the thrombocyte level lead this author to suggest that the effect of the anticoagulant *in vivo* must be local and confined to the intestinal lesions. The possibility of the existence of a toxin which effects the lowering of the hæmoglobin value is discussed and is considered by this author to be more probable than the existence of a toxic inhibition of the function of the bone marrow in this disease.

Sexual Sterilization in Alberta.

R. R. MACLEAN AND E. J. KIBBLEWHITE (*Canadian Public Health Journal*, December, 1937) state that in the past eight years, during which time sterilization has been legalized, 516 persons (181 males and 335 females) have been sterilized by operation. Mental deficiency, with or without psychosis or epilepsy, and psychosis constituted the chief bases for action. Consent was not obtained from 492 (320 men and 172 women) of the 1,008 patients recommended for sterilization. In Alberta a eugenics board of four, two of whom must be medical practitioners, may authorize the operation after presentation of the details of the case by at least two psychiatrists. Consent of the adult patient or, if the patient is a minor, of the parent or guardian, is then required, except in the case of mental deficients. Not only the risk of transmission of the defect, but the danger of the mental effect of procreation on the patient is considered. Consent was apparently more difficult to obtain from the male, the reason suggested being the blow to his pride or vanity. Operation, if performed at the official hospital, is carried out free of charge. Two post-operative deaths occurred, both in females, one due to bronchopneumonia and the other to general peritonitis. One male patient after operation impregnated his wife twice; at a second operation the vasa proved to be patent. The authors claim very satisfactory results from a social and psychiatric standpoint.

Hookworm Infestation.

J. ALLEN SCOTT (*The American Journal of Hygiene*, November, 1937) records an exhaustive study of hookworm infection in Egypt. His report comprises two million records from hospital out-patient departments and the direct examination of 40,000 specimens collected by the author in 125 villages. Of the twelve million rural inhabitants, five million are infected with hookworm. In the Fayum only 15% and north of the Delta only 20% of the population are infected, so that of the remaining nine millions about 50% are infected. The maximum degree of infestation appears to be reached by the female before the age of twenty, and by the male about the age of fifteen years. The infestation of the male is earlier, heavier and more persistent. Rate of infestation is, therefore, conspicuously associated with agricultural work. The type of crop or the methods of irrigation used did not appear to affect its prevalence.

Transitory Immunity to Yellow Fever in the Offspring of Immune Mothers.

F. L. SOPER, H. BEEUWKES, N. C. DAVIS AND J. A. KERR (*American Journal of Hygiene*, March, 1938) state that the presence of antibody in the serum at birth depends on its presence in the mother's serum. The immunity thus conferred is passive, rapidly disappearing during the first few months of life. It depends mainly on placental transference. The serum of all babies, whether their mothers are immune or not, contains no antibody to yellow fever after the babies are six months of age. Transmission of antibodies through the mother's milk cannot be entirely ruled out, but does not prevent the loss of immunity after six months of age.

British Medical Association News.

ANNUAL MEETING.

THE annual meeting of the Western Australian Branch of the British Medical Association was held at the Hospital for the Insane, Claremont, on March 20, 1938, DR. F. W. CARTER, the President, in the chair.

Financial Statement.

The honorary treasurer's report and balance sheet were adopted. The statement is published herewith.

Dr. T. C. Boyd and Dr. A. W. Farmer were reelected honorary auditors.

Annual Report of Council.

Dr. F. W. Carter read the annual report of the council. The report is as follows.

I have pleasure in presenting the report of council for the year ending March, 1938.

Membership.

The membership of the Branch has increased from 271 to 286.

Deaths.

It is with great regret that I have to report that four members died during the year: Dr. W. A. S. Bridgeford, of Boulder; Dr. Donald Cameron, of Collie; Dr. Fergusson Stewart, of Carnarvon; Dr. E. C. Dean, of Bendering.

General Meetings.

Nine general meetings were held, with an average attendance of 34, the largest at any meeting being 49. Year after year we expose the lack of interest displayed by members, as evidenced by the relatively small attendances at meetings of acknowledged merit and professional value. This year papers were read by Dr. Fortune, Dr. Jacobs, Dr. Hislop, Dr. Craig, Dr. G. C. Moss and Dr. Lucraft. They were all excellent papers, requiring painstaking preparation, and the average attendance of members was but 34. I join with a long line of retiring presidents in deploring this indifference of members and urging the necessity for improvement during the coming year.

The annual general meeting, March, 1937, was again held by the kind invitation of Dr. James Bentley at the Hospital for Insane, Claremont. A successful dinner was held at the Hotel Adelphi during post-graduate week, when 70 members were present.

Council Meetings.

Your Council held 11 meetings, members attending as follows:

Dr. Atkinson	6	Dr. Syme Johnson	5
Dr. Carter	11	Dr. Le Souef	11
Dr. Cuthbert	9	Dr. M. K. Moss	10
Dr. F. Gill	5	Dr. D. Smith	8
Dr. Hayward	11	Dr. Stewart	9

Federal representatives (by invitation)—

Dr. McWhae	4	Dr. Paton	8

A very large volume of work was transacted, first by 11 meetings of the executive and then by the council, much of which was first considered by the various subcommittees.

Federal Council.

Dr. McWhae and Dr. Paton were again appointed Federal representatives for 1938. Dr. Le Souef and the President attended the August meeting of the Federal Council, as Dr. Paton and Dr. McWhae were unable to attend, and the President acted in place of Dr. McWhae at the Federal meeting on February 10.

Australasian Medical Congress, 1940.

The invitation of this Branch for the holding of the 1840 congress in Perth has been accepted by the Federal Council, and the Senate of the University of Western Australia has kindly placed the use of the university building at our disposal during the September-October vacation in 1940. We are all very delighted that Dr. Paton has agreed to the council's wish for him to be president of the 1940 congress, and the Federal Council has confirmed this appointment. A very great amount of work lies before the congress committee during the next two years so as to assure that the congress will be a thorough success, and it is essential that every member give the committee loyal and hearty support.

Other Federal Matters.

Several other Federal Council matters have had the careful attention of your council.

Organization of the Medical Profession in Australia.—A suggestion emanating from the Federal Council was made during the year for a reorganization of the medical profession by the formation of an Australian Medical Association, with abandonment of the name of the Parent Body. This met with almost Australia-wide objection, and it was decided at the last meeting of the Federal Council to seek a greater measure of local autonomy as the best means of facilitating the work of the organized profession in Australia.

Uniform Medical Registration for Australia.—Your council is in favour of uniform medical registration in Australia, but is opposed to Federal control.

Civil Aviation Landing Grounds.—A sum of £10,000 has now been granted by Federal authorities.

Registration of Foreign Doctors who have found it necessary to leave Germany and who have obtained Diplomas after One Year's Clinical Study in Scotland.—Endeavours to have this matter rectified both by the Parent Body and by the General Medical Council of Great Britain having failed because of the unwillingness of the Conjoint Board of Scotland to abandon the registration of foreign graduates after one year's clinical study, the matter has now become one in which an amendment of the various State laws appears to be the only practical solution.

Recruitment of the Medical Profession in the Event of War, and Public Education and Control of Cancer.—These have received careful attention.

None of these items has reached the stage requiring approval of general meeting.

London Representative.

Dr. Isaac Jones has been again nominated by South Australia, Victoria, Tasmania and Western Australia for 1938-1940.

Alteration to Rules.

Three alterations have been made during the year: (i) increase in personnel of council from three to four; (ii) increase of metropolitan and country subscriptions by 10s. 6d. *per annum*; (iii) to provide for formation of special associations to be uniform with all Branches in Australia.

You will be asked tonight to confirm the alteration to rules to provide for members who have been members for fifty years or more to continue as members without paying subscription; this is to conform with a resolution of the Parent Body.

Maternal Mortality.

At the instigation of the Minister for Public Health, Mr. Munsie, the Government passed a bill in Parliament having for its object a magisterial inquiry into all deaths occurring as a result of childbirth. Your council was definitely against the proposal, but in view of the Minister's determination to carry out the oft-repeated promise made on the hustings, a special committee collaborated with him in the preparation of the bill. Both the nursing

and medical professions are represented on the Bench, and have equal powers with the other occupant, the local magistrate, and the preliminary inquiry will be made in camera. Since the act came into force various anomalies have been exposed, and your council has for the past few weeks been in communication with the Health Department and the Crown Law Department regarding the removal of these. It seems evident that the act will have to be amended in the very near future.

Hospital Policy.

A great deal of work has been done on this subject by subcommittees and your council. A deputation to the Minister for Health urged the appointment of a hospital commission to control all hospitals, consideration of which was promised. We secured British Medical Association and honorary staff representation on the committee to consider the Perth Hospital problem.

The matter of the shortage of resident medical officers was the subject of a deputation to the Under-Secretary of the Medical Department; but so far no improved conditions have resulted, although the Fremantle Hospital now has three resident medical officers.

On the question of staffing of private hospitals, recommendations have been made to the Commissioner of Public Health.

Your council was approached by the Department of Public Health with a view to ascertaining its views on the hospital policy of the country. This matter was fully considered and the department reminded of the undertaking given to a deputation from this council to the Minister for Health (Mr. Munsie) for the formation of a hospitals commission, which would have as its main function the hospital problem as a whole, and would be a permanent body to whom the Government might look for expert advice.

Professional Vigilance Committee.

All the professional bodies in Perth have joined together to form a professional vigilance committee to watch their interests. The council has supported this and appointed a representative.

The North-Western Aerial Medical Service has been formed in Western Australia into a corporate association, and your council has supported this and appointed the President for the time being your representative.

Ethics.

Many ethical rulings have been given, which include: professional rooms and name-plates in chemists' shops, advertising in the Press, authority to give lectures and addresses, life assurance certificates, pathologists and biochemists.

Statement of Receipts and Payments for Year ended December 31, 1937.

RECEIPTS.	£	s.	d.
January 1, 1937—			
Balance in Bank of New South Wales	539	15	8
Cash on Hand	14	14	0
Interest—			
Commonwealth Loans	101	18	10
Australasian Medical Publishing Company, Limited—Debentures	15	5	0
Annual Subscriptions	859	14	6
Anatomy School	5	5	0
Annual Dinner Fund	50	17	0
Collected for Medical Benevolent Fund	76	2	6
Collected for Medical Defence	0	10	0
	£1,664	2	6

PAYMENTS.	£	s.	d.
Printing, Stationery and Postages	116	17	7
Assistant Secretary's Salary (including Office Fee and Clerical Assistance)	150	0	0
Honorary Treasurer's Assistant's Salary	10	0	0
Legal Expenses	16	1	6
General Expenses	9	6	4
Library—			
Rent	6	0	0
Salaries	73	10	0
Books, Telephone *et cetera*	51	6	4
Subscriptions—			
Australian Aerial Medical Service	2	2	0
Professional Vigilance Committee	1	1	0
Taxes	12	16	6
London Account	303	16	6
Sydney Account	243	10	0
Commonwealth Loan	398	0	0
Refunds—			
Medical Defence	0	10	0
Medical Benevolent Fund	76	2	6
Dinner Fund Account	49	14	3
Balance at Bank of New South Wales, Perth, December 31, 1937	143	8	0
	£1,664	2	6

Invested Funds Account.

December 31, 1936—	£	s.	d.
Invested Funds on Hand—			
Commonwealth Bonds	2,510	0	0
Australasian Medical Publishing Company, Limited	345	0	0
Commonwealth Loan	400	0	0
	£3,255	0	0

December 31, 1937—	£	s.	d.
Invested Funds on Hand, held by Bank (as per certificate, January 5, 1938)—			
Commonwealth Treasury Bonds	2,910	0	0
Australasian Medical Publishing Company, Limited	345	0	0
	£3,255	0	0

(Signed) DONALD SMITH, Honorary Treasurer.
February 18, 1938.

(Signed) T. C. BOYD, Honorary Auditor.
(Signed) A. W. FARMER, Honorary Auditor.

Subcommittees.

Subcommittees have been formed to consider a standard mining agreement, chiropractice, and artificially induced abortions.

Model Lodge Agreement.

Conference has been held with the friendly societies *re* fractures.

April General Meeting.

Arrangements have been made to hold the April general meeting at Bunbury.

Diphtheria Immunization.

The council has represented to the Commissioner of Public Health its views on the duties of district medical officers and the provision of travelling expenses.

Infantile Paralysis.

Where parents of patients are unable to pay a fee to the medical specialist when called into consultation, your council has made a recommendation to the Commissioner of Public Health that a fee of one guinea should be paid for such consultation by the Government.

Workers' Compensation Act.

Unfair criticism of the profession during the debate on proposed amendments to the *Workers' Compensation Act* during the year has had the close attention of your council. A special subcommittee, known as the Medico-political Subcommittee, has been formed to watch your interests and to speak in the name of the profession when such action was deemed advisable. Apropos of this, members are requested to refrain from direct personal communication with the Press; such matters should be referred to this subcommittee.

At the request of your council, the Government Actuary has furnished a dissected statement of payments made to doctors as distinct from "other services" for a stated period; but a similar request to the insurance underwriters was refused. The statement obtained from the Government Actuary effectively disproved statements derogatory to the profession made by certain politicians during the debate already mentioned, and the refusal of the insurance underwriters to supply their figures leads to but one conclusion.

In view of the importance of this matter your council has again approached the Underwriters' Association restating the whole case and requesting the reconsideration of our request.

Office-Bearers.

The nominations for office-bearers this year were only sufficient to fill all positions, and therefore no ballot was necessary. I declare the following elected:

President: Dr. L. A. Hayward.
President-Elect: Dr. N. Cuthbert.
Ex-President: Dr. F. W. Carter.
Honorary Treasurer: Dr. Donald Smith.
Honorary Secretary: Dr. L. E. Le Souef.
Four Members of Council: Dr. J. Ainslie, Dr. F. Gill, Dr. M. K. Moss, Dr. H. Stewart.

I desire to place on record the valuable services rendered to this council and the Branch generally by our immediate Past-President, Dr. Syme Johnson, who has now retired from the administrative body. Dr. Syme Johnson, in addition to the presidential chair, occupied the position of honorary treasurer for four years and was a member of council, without portfolio, for three years. The Branch is deeply indebted to Dr. Johnson for many years of able counsel and valued opinion.

My year has been made a pleasant one by the generous advice and assistance received at the hands of the officers and members of council, with the honorary secretary, Dr. L. E. Le Souef, as willing as ever to subordinate personal convenience to council activities; and I thank them personally and in your name.

The thanks of this Branch are due to our lay secretary, Mr. Neilson Hancock, who, with his office staff, has carried out the duties of his appointment with characteristic efficiency and to the complete satisfaction of those whose duties brought them in close contact with him, often under conditions of hurry and stress. Your incoming committee will be faced with the urgent necessity for the provision of adequate board-room facilities for council, deputation and committee purposes. Such accommodation is long overdue, and with congress looming ahead it cannot further be postponed. Some such arrangement as the pooling of costs between ourselves, the Medical Board and our lay secretary, in agreed proportions, would seem to meet the case.

Induction of President.

Dr. F. W. Carter then introduced Dr. L. A. Hayward, the President for the ensuing twelve months.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held on November 17, 1937, at Saint Vincent's Hospital, Melbourne. The meeting took the form of a series of clinical demonstrations by members of the honorary medical staff of the hospital.

Neurological Surgery.

DR. F. MORGAN showed a number of patients upon whom he had operated for neurological conditions. A female patient, aged thirty-four years, whom he had seen first on January 20, 1937, had complained of pain in the neck with dragging of the right foot of seven years' duration. In 1933 she had been troubled with numbness in the right hand and arm, which had increased in severity for a year. After some X ray treatment, in 1934, there had been considerable diminution in the pain in the neck and in the disability of the right hand; but since then gradual loss of power and increase of stiffness of the legs had occurred, more evident on the right side than on the left. Following lumbar puncture one week before she came under notice, there had been a sudden increase in the loss of power of the right leg and left arm. On examination it was found that the gait was spastic, that the patient dragged the right foot, had moderate weakness of the neck muscles, especially those of flexion, and that she was unable to rise unaided from the recumbent position. The findings were those of spastic weakness of both upper and lower limbs, greater on the right side than on the left. The patient was barely able to write with the right hand, and was able to walk only about twenty yards in a very stiff fashion. The tendon reflexes of the upper and lower limbs were bilaterally increased, the abdominal reflexes were absent and the plantar reflexes were extensor in character. Bilateral ankle and patellar clonus was found, and the Rosollim reflexes were positive. Below the level of the clavicles there was some loss of sensation to cotton wool, to pin-prick, to heat and to cold, the greater loss being on the left side of the body and in the periphery rather than proximally; on the right side the loss extended above the clavicle into the trigeminal area. Dr. Morgan demonstrated in a skiagram of the cervical spine of the patient that there was an enlargement on the right side of the second intervertebral foramen to about four times the normal size. He said that the Queckenstedt test showed evidence of a partial block of the cerebro-spinal fluid pathway, and that the protein content of the fluid was 155 milligrammes per hundred cubic centimetres. The fluid looked clear, but two cells were found microscopically.

On February 2, 1937, Dr. Morgan performed cervical laminectomy, and a large neurofibroma was disclosed on the anterior surface of the cord in an extradural position. The tumour extended through the second right intervertebral foramen into the posterior triangle of the neck. He had been able to remove the tumour, and on the day following the operation increase in the power of the limbs was evident, the patient being able to lift the lower limbs

two feet from the bed, though before operation she had been able to lift them one foot only. Improvement had continued, and at the time of the meeting the patient was able to walk about in ordinary fashion, write well and do fine needle-work.

Another patient shown by Dr. Morgan was a man, aged thirty-four years, who had been referred to him on April 14, 1937, by Dr. Arthur Joyce, with a history of excessive alcoholism of four years' duration up to the year 1935. In April, 1937, the vision of the left eye had begun to fail, and in July, 1937, that of the right eye. The patient had been unable to read the newspaper since December, 1936, and the left eye had been blind since January, 1937. Throughout 1936 he had had attacks of diplopia, and for eight months he had had mild attacks of headache, readily relieved by acetylsalicylic acid. The patient had a moderate tendency to adiposity, pale, smooth, finely wrinkled skin, and genital hypoplasia. Bilateral primary optic atrophy was present; he could detect finger movements at two feet and the visual acuity was not more than $^{2}/_{36}$. Bitemporal hæmianopia for finger movements was demonstrable, and the pupillary reactions to light were sluggish, especially in the case of the right eye. In the skiagrams a greatly enlarged *sella turcica*, erosion of the anterior and posterior clinoid processes and partial obliteration of the sphenoidal sinus were visible.

On March 15, 1937, a large pituitary adenoma was disclosed through a left-sided transfrontal osteoplastic exposure. "Avertin" had been administered and local anæsthesia used. The tumour came away quite easily and was removed subtotally by suction and dissection. For four days following the operation the patient suffered from *delirium tremens*, and when he recovered from this state he was able to read the smallest print in the newspaper. When his sight was tested on June 16, 1937, the visual acuity on the left side was almost normal; and, apart from slight defects in the upper nasal quadrant and the peripheral temporal portion, the field of vision was normal. In the right eye the vision did not improve after the operation. It deteriorated somewhat, but the patient could still distinguish finger movements at a distance of three feet.

Dr. Morgan's third patient was a man, aged sixty-eight years, who had suffered from fits for thirty years. The first fit had occurred one month after he had suffered a severe blow on the head, which, however, had not rendered him unconscious. In a typical fit, following a peculiar sensation of the left arm going to the fingers, actual twitching of the left arm and fingers and of the left side of the face occurred, and later he became unconscious. The duration of the fit was usually about twenty minutes. Some of the fits occurred while he was walking about, and on these occasions he always fell to the left side. In the early stages the interval between the fits was six months or a year, but for nine or ten years they had occurred approximately every six weeks. On examination it was found that he had a paresis of the upper part of the face on the left side, which was ascribed to severance of the facial nerve that occurred when he cut the face in falling at the onset of one of the fits. Dr. Morgan had not seen him before November 16, 1937, but was able to demonstrate from skiagrams that there was an oval swelling about two centimetres long and one centimetre broad in the lower forward part of the right middle fossa of the skull. The appearance of the outline of the swelling suggested the presence of calcification in its margin, and the opinion had been expressed that the condition was due to an intracranial aneurysm.

Dr. Morgan also showed a female patient, aged thirty-four years, who, from the age of seven years, had had local fits commencing in the right side of the body with throbbing of the right side of the abdomen, numbness of the right side of the body, contracture of the right upper limb, drawing of the face to the left side and inability to speak. For twenty minutes after an attack she would have a feeling of weakness on the right side of the body. These attacks had occurred usually once or twice a week, but during the past eighteeen months they had been milder in degree. In June, 1937, she began to scrape the right foot along the ground and complained that the ankle felt cold. The weakness in the right lower limb had become progressively worse from March, 1937, at which time she had first noticed, in addition, that the right upper limb became fatigued very rapidly. Since March, 1937, she had suffered from severe bifrontal headaches, which were usually worse in the morning. When she came under his care on August 25, 1937, Dr. Morgan found that she had nystagmus on looking to the right, diplopia on looking upwards and to the right, and weakness of the lower part of the right side of the face on emotional movements. There were also moderate diminution of power of the right lower limb and increased tone in that limb at the ankle and knee, but there was no diminution in the power of the right upper limb, though its tone was increased in comparison with that of the left. The tendon reflexes of the right upper limb were slightly increased and the tendon jerks of the right lower limb were excessively active. The abdominal reflexes were diminished on the right side. The plantar reflexes were extensor in character on each side, but more definitely so on the left side than on the right side. Patellar and ankle clonus was present on the right side. The sense of position was lost in the great toe of the right foot, and there was the cortical type of loss of sensation for cotton wool and pin-prick over the right lower limb.

On September 5, 1937, Dr. Morgan turned a left lateral osteoplastic flap and disclosed the presence of a large cystic glioma in the upper portion of the left parietal lobe. Anteriorly the cyst extended forward beneath the motor cortex, and a large mural nodule occupied the upper portion of the superior wall of the cyst. The greater portion of the tumour was removed, but it had not been found possible to take all of it away because of its invasive nature at one portion of the circumference. Dr. Morgan stated that the patient had made a good recovery from the operation and had already regained her preoperative physical state, and there had not been any further fits. Microscopic sections of the tumour had been prepared and it was found to be an astrosarcoma; but, as there were anaplastic changes in the cells in one portion of the tumour, the patient was undergoing a course of deep X ray therapy.

The fourth patient shown by Dr. Morgan was a man, aged forty-nine years, whom he had first seen on February 2, 1937. In 1919 a ton of chaff had fallen on the patient's back, and immediately he had developed quadriplegia. Five weeks later he had been discharged from hospital, and after another five weeks he had resumed work. In 1923 pains developed in both legs, with loss of power of the feet, incontinence of fæces and retention of urine. After spending six months in hospital he was able to work again; the bowel and bladder were normal, but he still had some numbness in his feet. He led a more or less normal life until early in 1934, when he began to suffer as in 1923, with retention of urine, incontinence of fæces and pains in the legs. Between 1934 and 1937 he catheterized his own bladder. On examination in February, 1937, the tendon jerks of the upper limb were greatly increased in activity, the abdominal reflexes were absent, the knee jerks were excessively active, the ankle reflexes were absent and the plantar reflexes were absent. There was gross wasting of all the muscles below the knees on both sides; the feet were clawed and there was considerable wasting of muscles of the buttocks and of the posterior aspects of the thighs. Fibrillation was present in the wasted muscles. The patient had retention of urine and paralysis of the sphincter muscle of the bowel, and there was loss of sensation for cotton wool and pin-prick in the fifth lumbar segments and in all the sacral segments. Dr. Morgan considered it probable that a double lesion was present, affecting the cervical cord and the *cauda equina* as the result of the injury in 1919.

On February 12, 1937, a Queckenstedt test was performed with the needle in the interspace between the fourth and fifth lumbar vertebræ; the resting pressure of the cerebrospinal fluid was 120 millimetres of water, but bilateral jugular compression brought about a rapid rise to 500 millimetres on the manometer, with a rapid fall on release of the compression. On February 24, 1937, 0·75 cubic centimetre of "descending" lipiodol was injected into the spinal theca though the interspace between the second and third lumbar vertebræ; subsequent radiography established

the presence of a complete block to the passage of the lipiodol. The lower border of the lipiodol was held up at the lower border of the third lumbar vertebra.

On April 29, 1937, Dr. Morgan performed lumbar laminectomy, the laminæ of the lumbar vertebræ being removed. These laminæ were dense like ivory and were twice the normal thickness. When they were removed and the *dura mater* was incised the roots of the *cauda equina* were found to be bound firmly together by fibrous tissue. They were separated in the mid-line and a small tumour, about twice the size of a pea, was disclosed lying in the middle line and in front of the anterior layer of dura. It was easily removed. It proved to be an extruded portion of the intervertebral disk. Dr. Morgan said that within a few weeks of the operation sensation had begun to return to the anal canal, but there was no improvement in the condition of the bladder; to relieve this, permanent suprapubic drainage had been instituted. The patient had informed him that the pain in the legs had gone, and that though it was still not very good, walking had become much easier.

Diabetes Mellitus Treated with Protamine Zinc Insulin.

DR. T. A. F. HEALE showed a woman, aged twenty-six years, who had *diabetes mellitus* and who had been treated with protamine zinc insulin. Typical diabetic symptoms—thirst, polyuria, loss of weight and strength, and *pruritus vulvæ*—developed towards the end of June, 1937. When the patient was examined on August 3, 1937, the urine contained a trace of ketones and gross glycosuria was present. The blood sugar estimation was 0·31 milligramme per 100 cubic centimetres four hours after a meal. The patient's usual weight was said to be 55·0 kilograms (eight stone seven pounds), but on that day it was 46·7 kilograms (seven stone five and three-quarter pounds). No other abnormal findings were recorded on general examination and there was no family history of diabetes.

The patient was admitted to the ward on August 7, 1937. The initial diet was composed of 156 grammes of carbohydrate, 71 grammes of protein and 84 grammes of fat, and was estimated to contain 1,664 calories. Thirty-two units of protamine zinc insulin and 16 units of regular insulin were given fifteen minutes before breakfast, and twelve units of regular insulin were given fifteen minutes before the evening meal. On the third day the regular insulin before the evening meal was omitted. By August 18, 1937, the dose of protamine zinc insulin had been reduced to twenty-eight units and that of regular insulin to eight units. The diet was increased to 173·5 grammes of carbohydrate, 72 grammes of protein, and 87 grammes of fat, the caloric value being 1,765 calories. The urine tests revealed much less sugar and the fasting blood sugar was 0·09 milligramme per 100 cubic centimetres. By August 31, 1937, by the use of this diet, with twenty-four units of protamine zinc insulin and six units of regular insulin, the urine was usually sugar-free throughout the day. On August 31, eight estimations were made of the blood sugar content at intervals of two or three hours; the lowest estimation was 0·03 milligramme per 100 cubic centimetres at 10.30 a.m. and again at 4.30 p.m., and the highest was 0·14 milligramme per 100 cubic centimetres at 4.30 p.m. In spite of the low blood sugar values the patient did not show any symptoms of insulin shock. The regular insulin was discontinued, and on September 3 the patient was discharged from hospital, with directions to continue the diet and to have twenty-four units of protamine zinc insulin before breakfast. At the time of the meeting the patient was very well and had resumed work as a forewoman in a knitting mill. Her weight had increased to 55·35 kilograms (eight stone eleven pounds). The urine was usually sugar-free, though occasionally an "orange" reaction occurred. The fasting blood sugar level was 0·11 milligramme per 100 cubic centimetres, and twenty-four units of protamine zinc insulin were still being injected fifteen minutes before breakfast. The diet had not been changed.

Dr. Heale said that he considered that a very satisfactory result had been obtained with protamine zinc insulin in this case. He had used this method of treatment twelve times only, but he intended to use it more and more. Two-thirds of the patients were well under control by means of it; only one of his results could be classed as very unsatisfactory, but the condition of this patient had not been well controlled at any time on any plan of diet and regular insulin. In half the cases protamine zinc insulin had been used alone and in the others regular insulin had been used in addition. The injections of insulin had been given thirty to fifteeen minutes before breakfast. Dr. Heale said that he was in the habit of advising a small supper about 9.30 p.m. consisting of ten to twenty grammes, and he often gave small amounts of carbohydrate, about ten grammes, at morning tea and afternoon tea. He said that the reports of the use of protamine zinc insulin in other countries, though somewhat contradictory, indicated that it represented a distinct advance in treatment, even in the present incomplete state of knowledge of how best to use it. In a recent report of its use in a series of over 1,250 patients, in 50% of cases in combination with regular insulin, Joslin had classified control as good in 70% of the cases, and fair in the remainder, with the exception of about 2% in which the control was bad. Dr. Heale considered that patients who were doing well on regular insulin should not be transferred to protamine zinc insulin unless the fasting blood sugar values were constantly high, but that new patients requiring insulin might be treated suitably with the new insulin. Patients who were difficult to control with regular insulin were likely to be difficult to control with the new insulin, which would not solve all the difficulties. In conclusion, Dr. Heale said that anyone who wished to use the new insulin should be well acquainted with its properties and with the plans recommended, as the technique of treatment differed considerably from that with regular insulin. He thought that the new insulin would probably become the insulin of choice, though he considered that regular insulin would continue to have a place in treatment.

DR. J. HAYDEN showed a male patient, aged fifty-four years, suffering from *diabetes mellitus*, which had been stabilized by the use of protamine zinc insulin. The patient had required thirty-five units of ordinary insulin daily; but at the time of the meeting the urine was sugar-free and the patient was receiving twenty units of protamine zinc insulin in one injection given before breakfast each day. There had been no insulin reaction. Dr. Hayden drew attention to an unusual feature of the case. A complete peroneal nerve lesion had occurred suddenly, in 1933, while the patient was sitting in a chair reading. Three months earlier he had had an accident in which a weight had fallen on his back, and a fissure had been found in the fifth lumbar vertebra. He had been treated in hospital after the accident and had [illegible] ently rested for two months. Dr. Hayden did [illegible] possible to correlate the peroneal nerve lesion [illegible] accident. At the time the nerve lesion had occurred the patient was not wearing any splints or bandages. The lesion was regarded as due to a thrombosis in the vessels supplying the peroneal nerve. No recovery from the lesion had occurred in the intervening three years.

Hypertension and Cerebral Hæmorrhage.

Dr. T. A. F. Heale showed a female patient, aged twenty-four years, who had been admitted to the hospital on October 1, 1937. Five weeks earlier, after previous good health, the onset of the present illness had been characterized by repeated vomiting for three days without headache. After one week in bed, severe frontal and occipital headache had commenced when she got up, aggravated by movement, and pain which also radiated down the back of the neck. One week before her admission to hospital, she had developed a particularly severe headache, her gait had become unsteady and her behaviour had caused people to wonder whether she had been drinking. She returned to bed and became drowsy, and when seen by her own doctor late that day she was still drowsy. He found a left hemiparesis, and the left plantar reflex was extensor in character. The patient was still drowsy on admission to the hospital, but could be roused easily and could talk rationally though slowly, and she complained of headache. The systolic blood pressure was 240 and the diastolic 165 millimetres of mercury. There was no evidence of enlarge-

ment of the heart, and the sounds were regular and clear, though the second aortic sound was accentuated. The radial arteries were slightly but definitely thickened. No neck stiffness was present. The cranial nerves were normal, with the exception of slight weakness of the left facial nerve. Power and sensation were normal. The deep reflexes were equally active. The right plantar reflex was flexor and the left one was equivocal. Uniformly blood-stained fluid was obtained on lumbar puncture. The blood serum and cerebro-spinal fluid failed to react to the Wassermann test; the results of blood urea and urea concentration tests were normal. The urine did not contain albumin or sugar, and no abnormality was detected microscopically. Radiographically, it was considered that the heart was slightly enlarged. Some small white areas were detected in the macular region of the left eye, but in other respects the fundi were normal in appearance. The patient became less drowsy and the headache was greatly relieved by the lumbar puncture.

Dr. Heale said that at the time of the meeting the patient was free from symptoms and felt very well, but the blood pressure was still very high and very slight left-sided facial paresis was present. He said that he had shown the patient because cerebral hæmorrhage at that age was distinctly uncommon, though hypertension was not so uncommon. There seemed to be no doubt that she had had a subarachnoid hæmorrhage, though it was not easy to explain the hemiparesis. The cause of the hypertension had not been determined. There was not any evidence of chronic nephritis or of coarctation of the aorta. He discussed the possibility of the presence of a suprarenal tumour, which he considered unlikely because of the persistent hypertension. Tumours of the adrenal glands and medulla and related structures were likely to be characterized by paroxysmal attacks of hypertension rather than by long-continued elevation of blood pressure. He concluded that the condition could be classified as an example of the so-called essential vascular hypertension, and discussed the various surgical procedures which had been advocated; but he stated that he did not contemplate surgical treatment for the patient at that time.

Aortic Stenosis.

DR. C. FITTS showed a series of patients to demonstrate some of the problems of ætiology and of symptomatology in aortic stenosis. He said that he considered that establishment of the presence of the pulse features which were usually regarded as diagnostic of aortic stenosis was of no more than academic importance. He thought that it was far easier to determine the existence of a thrill accompanying an aortic systolic murmur, and that the presence of a thrill had greater significance than that of the characteristic pulse if it accompanied an aortic systolic murmur, whether this was regarded as indicative of stenosis or not. Each patient having a loud aortic systolic murmur should in his opinion be examined with very light palpation in full expiration, sitting up and leaning forward. There were ten patients in attendance at his out-patient clinic in whom a thrill could be felt; the condition could not be rare, and it was probable that it was not found more frequently because it was not sought. He thought of these patients as the subjects of aortic stenosis, and did not see that it was incompatible with even a high degree of aortic incompetence, sufficient to validate the test of the pulse though the thrill would remain. He believed that these opinions corresponded with the view that was adopted abroad, and that they were capable of confirmation at autopsy.

Dr. Fitts showed seven patients who had in common the presence of a systolic murmur and a systolic thrill at the aortic area, to demonstate that whatever meaning was attached to the syndrome the histories tended to run true to type. He stated that in five out of the seven cases the presence of calcified aortic valves had been established by fluoroscopic examination. He divided the seven patients into three groups.

The first group consisted of three patients with a definite rheumatic history. They showed clinical and radiological evidence of mitral stenosis, and in Dr. Fitts's opinion also had aortic valvular stenosis and incompetence. He emphasized that his main reason for stressing the presence of aortic stenosis in these patients was not that he valued the signs in assessing prognosis, but in order to illustrate that in these patients with a rheumatic history there was a considerable degree of aortic incompetence as well as mitral valvular disease.

In the second group Dr. Fitts showed two men, both in the early fifth decade, whose presenting feature had been angina of effort. In one patient it had appeared at the age of thirty-two years, and in each case had continued over a period of years. Neither of the patients had a rheumatic history, and in both patients, in addition to the common features quoted, there was an absence of the second sound at the aortic area and no diastolic blow could be found down the left border of the sternum; moreover, there was no evidence of involvement of the mitral valve. In each case inversion of the *T* wave in leads I and II was demonstrable in the electrocardiogram; and the blood serum had failed to yield the Wassermann reaction.

Dr. Fitts mentioned that he had two other patients in the clinic at that time who could be included in that group. Cabot had somewhat dogmatically ascribed the condition to rheumatism, but Dr. Fitts thought that the evidence was very slender. Dr. Fitts contrasted them with the former group, in which there was evidence of aortic incompetence and of mitral stenosis, but a most significant absence of anginal pain. The changes might possibly be due to early degeneration and calcification, with associated atheroma, of the coronary vessels. In one of the patients he had shown that opinion had been upheld by the Repatriation Board, and the man had been granted a full pension, though he had been repeatedly refused a pension before that view was placed before the board.

The third group shown by Dr. Fitts was made up of two female patients, whose signs and symptoms conformed to those of the type accepted as due to degenerative changes occurring in later life.

Coronary Occlusion.

DR. E. COOPER showed two male patients who had had acute coronary thrombosis at an earlier date, with recovery. One man, aged fifty-six years, had had the attack in March, 1935, and at the time it had been diagnosed as bronchopneumonia. The onset was associated with pain in the left side of the chest and moist sounds at the bases, and the temperature had been elevated to 38·9° C. (102° F.) for three days. The systolic blood pressure was 130 and the diastolic pressure 90 millimetres of mercury. A trace of sugar was found in the urine. The patient was in a condition of shock and had the typical electrocardiographic changes. At the time of the meeting the patient was still subject to substernal pain on exertion, which was relieved by the administration of nitrites; and the left side of the heart was enlarged. The systolic blood pressure was 190 and the diastolic pressure was 120 millimetres of mercury. He was able to work, was not dyspnœic and could lift heavy weights.

The other patient shown by Dr. Cooper was fifty-two years of age; the onset of the condition in his case occurred in April, 1937, and was characterized by collapse and severe substernal pain. On his admission to hospital the systolic blood pressure was 100 and the diastolic pressure 80 millimetres of mercury. After two months' rest in bed he was discharged from hospital, and at that time the systolic blood pressure was 120 and the diastolic 95 millimetres of mercury. At the time of the meeting he was able to carry out a full day's work in a clerical position without discomfort, though he became dyspnœic on exertion. He had not had any pain or œdema, and there was no demonstrable cardiac enlargement. The electrocardiogram shown illustrated the changes typical of the condition. The systolic blood pressure was 125 and the diastolic pressure 90 millimetres of mercury.

Dr. Cooper said that at Saint Vincent's Hospital 117 patients had been admitted to the wards since 1930 with the diagnosis of coronary occlusion. Thirty-two had died in hospital, a mortality of 27%, and 85 (73%) had left the hospital alive and had been discharged to the out-patient department or to a home. In 1928 Parkinson and

Bedford had stated that a patient seen alive with coronary thrombosis was more likely to survive the attack than to succumb to it. In 1930 Connor and Holt had reached the conclusion that the immediate mortality of the first attack of coronary thrombosis was 16·2% in the series of cases they had analysed. In 1935 Bedford had made the statement that in general a patient had at least a three-to-one chance of surviving the attack of coronary thrombosis. The statistics submitted by Dr. Cooper illustrated Bedford's statement closely.

DR. T. E. LOWE demonstrated from some experimental work in connexion with the distribution of the scars of coronary infarctions. Hearts injected with coloured gelatin preparations showed good capillary injections, and it was possible to find what areas were supplied by various branches of the coronary vessels. Dr. Lowe also showed some illustrations of the reconstruction of these myocardial scars.

(To be continued.)

NOMINATIONS AND ELECTIONS.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Rawle, Ronald Maxwell, M.B., B.S., 1930 (Univ. Sydney), 143, Macquarie Street, Sydney.

Dawson, Elsie Joan, M.B., 1922 (Univ. Sydney), 133, De Boos Street, Temora.

Atkins, Walter Thomas Garner, M.B., B.S., 1937 (Univ. Sydney), Kildare Street, Blacktown.

The undermentioned has applied for election as a member of the Victorian Branch of the British Medical Association:

Dobbin, Alexander Hamilton, L.R.C.P. & S. (Edinburgh), L.R.F.P.S. (Glasgow), Werribee.

The undermentioned have been elected members of the Victorian Branch of the British Medical Association:

Morris, Maurice, M.B., B.S., 1938 (Univ. Melbourne), Moore Street, Moe.

Hardy, Lowen Alexander, M.B., B.S., 1934 (Univ. Melbourne), Flinders Naval Depot, Crib Point.

Beck, Geoffrey Frederick, M.B., B.S., 1935 (Univ. Melbourne), c.o. Dr. Rait, Pakington Street, Geelong.

Hills, Stewart, M.B., Ch.B., 1918 (Univ. Melbourne), 712, Sturt Street, Ballarat.

Hughes, James Francis, M.B., B.S., 1936 (Univ. Melbourne), Austin Hospital, Heidelberg.

Post-Graduate Work.

WEEK-END COURSE IN ELECTROCARDIOGRAPHY IN SYDNEY.

THE New South Wales Post-Graduate Committee in Medicine will hold a course of instruction in electrocardiography during the week-end July 2 and 3, 1938. The course will be elementary and is particularly designed for those in general practice. The programme is as follows.

Saturday, July 2: Royal Prince Alfred Hospital.

2 p.m. to 2.30 p.m.—(i) "Principles of the Electrocardiograph", (ii) "The Modern Cardiograph", (iii) "The Normal Cardiogram": Dr. J. K. Maddox.

2.45 p.m. to 3.15 p.m.—"Arrhythmias", Part I: Dr. J. Halliday.

3.30 p.m. to 4 p.m.—"Arrhythmias", Part II: Dr. T. M. Greenaway.

4 p.m. to 4.15 p.m.—Practice in reading tracings of the above type.

Sunday, July 3: Sydney Hospital.

10 a.m. to 10.45 a.m.—"The Cardiogram in Coronary Disease", Dr. A. J. Hood Stobo.

11 a.m. to 11.30 a.m.—"The Cardiogram in Rheumatic, Thyreoid and Syphilitic Heart Disease and in the Acute Fevers": Dr. Wilfred Evans.

11.30 a.m. to 12.30 p.m.—General discussion and practice in reading cardiograms, under supervision.

The course will not be held unless eight applications are received before Thursday, June 30, 1938. The fee will be one guinea. Those wishing to attend should apply to the Secretary, New South Wales Post-Graduate Committee in Medicine, the University of Sydney, Sydney.

Correspondence.

NATIONAL HEALTH INSURANCE.

SIR: As an average general practitioner member of the profession, one perhaps not so enthusiastic and altruistic as Dr. Boyd (THE MEDICAL JOURNAL OF AUSTRALIA, May 14, 1938), nor yet so devoid of consideration for the patient as Dr. Marshall believes the species (THE MEDICAL JOURNAL OF AUSTRALIA, May 7, 1938), I should like to make a few comments on the proposed national health insurance scheme and capitation rates.

The leading article of THE MEDICAL JOURNAL OF AUSTRALIA (May 14, 1938) gives an explanation of the proposed agreement and capitation fee. In arriving at the capitation fee our delegates to the conference with the Federal Government have obviously assumed that the existing friendly society rates of remuneration are wholly satisfactory to the general practitioners of Australia, and that a compulsory extension at the same rates to the whole of the salaried population with an income below £365 *per annum* would be acceptable. In common with the vast majority of those members of the profession whom this insurance scheme will affect, I cannot agree. Whereas we may agree that these rates have been sufficiently high for a member earning £4 or £5 per week, we do not agree that a person earning £7 per week is entitled to the same benefits at the same rates. A fact that has been lost sight of is that the bulk of our private practice work, as distinct from contract work, has come from persons in the income field between £250 and £365. We consider that the average person in this field returns us more as a private patient than he or she would do as a compulsorily insured person on the basis of the present friendly society rates. For this reason we have agreed upon £1 as a fair and reasonable capitation rate if the Government desires to remove persons in this field from the sphere of private practice. A great number of our local associations, composed almost entirely of general practitioners, passed resolutions to this effect and had their delegates to conference agree upon this figure. They are now dismayed at the apparent complete indifference to their interests shown by those they trusted to safeguard them. How any delegates could feel themselves free to agree to terms so vastly different from those they were instructed to secure, without reference to those they were representing, passes comprehension and, we feel, merits strong condemnation. We do not need to be told that the Government representatives would raise all sorts of arguments to keep the rate down. The evident fact to us is that we requested a certain fee as the price of our cooperation in the Government scheme. Since it was not an extortionate fee, representing only about 4½d. per week from a person probably averaging 10s. per week on amusement, we feel that we have been dealt with in a niggardly way and that no terms so unacceptable should be agreed to. The proposed rates of remuneration, exchange rates and cost of living taken into consideration, are below those ruling in England. Are the Australian general practitioners less worthy of consideration than their fellows in England?

If we once accept unsatisfactory terms, let us not be foolish enough to expect a later readjustment; witness the recent failure of such an attempt in England.

It seems, then, that the time is overdue for us to take a definite self-protecting stand and demand now a just capitation rate as the price of our cooperation in a scheme that will affect us so vitally and which cannot exist without us. Each man's complete loyalty to his local association in any decisions taken is of vital importance. Let the general practitioners actively and quickly organize and refuse to work under a scheme whose rates and conditions are so unacceptable to the majority. It is a matter of almost life and death to the general practitioner sections of the profession. Let us use our power to retain life!

Yours, etc.,

K. F. TARLINTON.

Murwillumbah,
New South Wales,
May 18, 1938.

SIR: Your leader of the 14th instant has been the subject of considerable discussion by members of this local association. The lay Press has taken it to be an authoritative reply of the British Medical Association to criticism by members, and use has been made of it to stampede medical practitioners into accepting an agreement with which they are not in complete accord.

This local association feels that it is a dangerous principle for a medical publication to take upon itself the right and the authority of admonishing the profession as a whole and attempting to direct its footsteps in a predetermined direction.

Some other interesting facts come to light upon a close study and comparison of your leader and the statement published on April 16. Then it was inhalant anæsthetics which were excluded from the scheme; now it is an anæsthetic requiring the presence of a second practitioner. Also, irrespective of the type of anæsthetic and the nature of the operation, the maximum fee that can be charged an insured person is the sum of one guinea. It is also now stated for the first time that after an anæsthetic has been administered to an insured person a report has to be made within ten days to the local officer of the commission. This is, of course, the introduction into Australia of one of the worst features of the British scheme—unnecessary certification and unnecessary writing of reports. This disclosure leads us to ask if there are any other features of the agreement which have not been published. We think that, in fairness to the profession, the full text of the decisions arrived at by the executive committee of the Federal Council in their conference with representatives of the Federal Government should now be published so that we may know exactly where we are being led.

Finally, sir, we would like to ask if it is the policy of the British Medical Association that the friendly societies should be recognized as the sole approved societies.

Yours, etc.,

G. RUSSELL,
Honorary Secretary,
Canterbury-Bankstown
Medical Association.

Earlwood,
New South Wales,
May 21, 1938.

BLOTTING-PAPER ADVERTISEMENTS.

SIR: Might I appeal to the various drug companies who are in the habit of enclosing blotting-paper advertisements of their goods to refrain from doing so. It is most annoying to find one's books and records blotched and smudged through using this cheap blotting-paper. Another aggravating example today has at last forced me to do what I have been intending to do for several years past, namely, to write this letter, which I earnestly trust will have the desired effect. Some of us are bad enough writers without having our notes hopelessly smudged by cheap blotting-paper.

Yours, etc.,

"LONG SUFFERING."

Perth,
Western Australia,
May 17, 1938.

A DISCLAIMER.

SIR: Might I request sufficient of your space to disclaim any responsibility for an article appearing in the current issue of *Smith's Weekly*, which purports to describe my treatment of cases of schizophrenia by "Cardiazol". This article is quite misleading in its reference to the insulin hypoglycæmic treatment of schizophrenia and misrepresents the work being done by myself and others with "Cardiazol".

Yours, etc.,

REG. S. ELLERY.

33, Collins Street,
Melbourne, C.1,
May 22, 1938.

Proceedings of the Australian Medical Boards.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts, 1925 to 1933*, of Queensland, as duly qualified medical practitioners.

Bracken, Desmond John Gordon, M.B., B.S., 1936 (Univ. Sydney), Pomona.

Coles, Jack Lewis, M.B., 1936 (Univ. Sydney), Townsville.

Fitzpatrick, Eugene Francis, M.B., B.S., 1935 (Univ. Melbourne), Brisbane.

Goldmann, Hans, L.R.C.P. and S. (Edinburgh), L.R.F.P. and S. (Glasgow), 1937, Brisbane.

SOUTH AUSTRALIA.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Practitioners Act of* 1919, of South Australia, as duly qualified medical practitioners:

Turner, Robert Henry Alister, M.R.C.S. (England), L.R.C.P. (London), 1934, Morgan.

Schafer, David Paul Hannaford, M.R.C.S. (England), L.R.C.P. (London), 1927, M.B., B.S., 1930 (Univ. Melbourne), M.R.C.P. (London), 1932, Mental Hospital, Parkside.

Obituary.

DONALD STEWART MacCOLL.

WE regret to announce the death of Dr. Donald Stewart MacColl, which occurred on May 21, 1938, at Camberwell, Victoria.

NICHOLAS CLEMENT LARKINS.

WE regret to announce the death of Dr. Nicholas Clement Larkins, which occurred recently at Marrickville, New South Wales.

Books Received.

THE ESSENTIALS OF MATERIA MEDICA, PHARMACOLOGY AND THERAPEUTICS, by R. H. Micks, M.D., F.R.C.P.I.; Second Edition; 1938. London: J. and A. Churchill Limited. Large crown 8vo, pp. 390. Price: 12s. 6d. net.

THE PRACTICE OF REFRACTION, by Sir Stewart Duke-Elder, M.A., D.Sc., Ph.D., M.D., Ch.B., F.R.C.S.; Third Edition; 1938. London: J. and A. Churchill Limited. Large crown 8vo, pp. 376, with illustrations. Price: 12s. 6d. net.

MODERN TREATMENT IN GENERAL PRACTICE: A YEAR-BOOK OF DIAGNOSIS AND TREATMENT FOR THE GENERAL PRACTITIONER, edited by C. P. G. Wakeley, D.Sc., F.R.C.S., F.R.S.E., F.A.C.S.; Volume IV; 1938. London: The Medical Press and Circular. Demy 8vo, pp. 451, with 113 illustrations. Price: 10s. 6d. net.

A TEXT-BOOK OF X-RAY DIAGNOSIS, by British Authors, edited by S. C. Shanks, M.D., P. Kerley, M.D., M.R.C.P., D.M.R.E., and E. W. Twining, M.R.C.S., L.R.C.P., D.M.R.E.; Volume II. Crown 4to, pp. 469, with 307 illustrations. Price: 42s. net.

A METHOD OF ANATOMY, DESCRIPTIVE AND DEDUCTIVE, by J. C. B. Grant, M.C., M.B., Ch.B., F.R.C.S.; 1937. London: Baillière, Tindall and Cox. Crown 4to, pp. 670, with 564 illustrations. Price: 27s. net.

Diary for the Month.

JUNE 7.—New South Wales Branch, B.M.A.: Organization and Science Committee.
JUNE 10.—Queensland Branch, B.M.A.: Council.
JUNE 14.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JUNE 15.—Western Australian Branch, B.M.A.: Branch.
JUNE 21.—New South Wales Branch, B.M.A.: Ethics Committee.
JUNE 22.—Victorian Branch, B.M.A.: Council.
JUNE 23.—New South Wales Branch, B.M.A.: Clinical Meeting.
JUNE 24.—Queensland Branch, B.M.A.: Council.
JUNE 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JUNE 30.—South Australian Branch, B.M.A.: Branch.
JUNE 30.—New South Wales Branch, B.M.A.: Branch.

Medical Appointments.

Dr. J. F. F. Drew has been appointed, under the provisions of the *Quarantine Act*, 1908-1924, Quarantine Officer at Cossack, Western Australia.

* * *

Dr. W. C. Sangster has been appointed Honorary Officer of the Second Ear, Nose and Throat Clinic at the Adelaide Hospital, Adelaide, South Australia.

* * *

Dr. T. M. Gilbert has been appointed District Medical Officer at Cue, Western Australia, pursuant to the provisions of *The Hospitals Act*, 1927, of Western Australia.

* * *

Dr. J. F. F. Drew and Dr. R. L. Fulton have been appointed Medical Inspectors of Seamen at Cossack, Western Australia and Geelong, Victoria, respectively, in accordance with the provisions of the *Navigation Act of* 1912-1935.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xxii to xxiv.

FREMANTLE HOSPITAL, FREMANTLE, WESTERN AUSTRALIA: Medical Superintendent.
THE BAKER INSTITUTE, MELBOURNE, VICTORIA: Director.
THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY, NEW SOUTH WALES: Resident Medical Officers.
VICTORIAN EYE AND EAR HOSPITAL, MELBOURNE, VICTORIA: Resident Surgeons.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognise any claim arising out of non-receipt of journals unless such a notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.